THE PATHOLOGY OF THE EYE

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CHAPTER XXVIII

ABBREVIATIONS

A. d'O.—Archives d'Ophthalmologie.

A. f. A.—Knapp and Schweigger's Archiv für Augenheilkunde. (Articles in A. f. A. are often translated or abstracted in A. of O., and *vice versâ ;* the reference is usually given to one only.)

A. f. O.—v. Graefe's Archiv für Ophthalmologie.

A. of O.—Knapp's Archives of Ophthalmology.

B. d. o. G.—Bericht der ophthalmologische Gesellschaft zu Heidelberg. (The earlier reports are contained in K. M. f. A.)

B. z. A.—Deutschmann's Beiträge zur Augenheilkunde. (The reference is given to the part [Heft], not to the volume.)

C. f. A.—Hirschberg's Centralblatt für praktische Augenheilkunde.

G.-S.—Graefe-Saemisch, Handbuch der gesamten Augenheilkunde. (The date determines the edition : 1st edition, 1874—1877 ; 2nd edition, 1898—   .)

K. M. f. A.—Zehender's Klinische Monatsblätter für Augenheilkunde.

R. L. O. H. Rep.—Royal London Ophthalmic Hospital Reports.

T. Am. O. S.—Transactions of the American Ophthalmological Society.

T. O. S.—Transactions of the Ophthalmological Society of the United Kingdom.

Z. f. A.—Zeitschrift für Augenheilkunde.

*.—The most important articles are marked with an asterisk (*).

ERRATUM

On p. 1248, line 15 from top, for "mentioned" read "foreshadowed."

CHAPTER XXII

INJURIES OF THE EYE

Injuries of the eye may be classified thus :

I. Superficial injuries without penetration of the globe caused by sharp or blunt instruments, and injuries by the action of heat, chemical caustics, electricity, etc.

II. Injuries by blunt objects.

III. Penetrating injuries without retention of a foreign body within the globe.

IV. Penetrating injuries with retention of a foreign body within the globe.

V. Displacement of the eye as a whole.

VI. Injuries of the optic nerve.

VII. Injuries of the orbit.

The text-books of Bartisch, St. Yves, Taylor, Scarpa, Beer, Stellwag, Mackenzie, Desmarres, v. Arlt, and others. White Cooper.—On Wounds and Injuries of the Eye, London, 1859. Zander and Geissler.—Ueber die Verletzungen des Auges, Leipzig and Heidelberg, 1864. George Lawson.—Injuries of the Eye, Orbit, and Eyelids, London, 1867. v. Arlt.—Ueber die Verletzungen des Auges mit besonderer Rücksicht auf deren gerichtsärztliche Würdigung, Wien, 1875. Yvert.—Traité practique et clinique des Blessures du Globe de l'Œil, Paris, 1880. *Praun.—Die Verletzungen des Auges, Wiesbaden, 1899. Baudry.—Étude medico-legale sur les Traumatismes de l'Œil, Paris, 1903. Roselli.—Traumatismi oculari, 1903. Beaumont.—Injuries of the Eyes of the Employed and the Workmen's Compensation Act, London, 1907.

I. Superficial Injuries, Injuries by Heat, Electricity, etc.

Abrasions of the cornea by minute foreign bodies are probably of everyday occurrence. Their chief danger lies in the removal of the epithelium, the first line of defence against bacterial invasion, particularly when a mucocele is present. Corneal abrasions leave no scar unless Bowman's membrane has been penetrated and destroyed. Injuries with twigs of trees, leaves of plants, and particles of stone show a special tendency to give rise to hypopyon ulcers, not satisfactorily explained by the occasional presence of lacrymal obstruction (Hillemanns).

Some erosions of the cornea, especially those caused by the finger-nail, show a tendency to give rise to recurrent irritation—traumatic keratalgia, cicatrix dolorosa, etc. (v. Arlt, Cooper, Mooren, Szili, Grandclément, Markwort, Eliasberg, Bronner, Fuchs, Salzmann,

Nieden, Hirsch, Praun). It is probable that the regeneration of the epithelium is defective, so that vesicular keratitis is readily induced and leads to fresh denudation (Fuchs, Salzmann, Stood).

Haltenhoff described ulceration resembling keratitis dendritica following a blow with a cow's tail. Severe and chronic keratitis results from abrasions contaminated with vaccine, such as have been caused by a lancet or by particles of a broken vaccine tube (Critchett, Sénut, Hirschberg, Schmitz, Schirmer, and others). Staining of the cornea may be caused by injuries with ink and inky pens (Bergmeister, Mayerhausen, Haass).

The influence of injury as an exciting agent of interstitial keratitis must be borne in mind (Bietti, Bronner, Armaignac, Csapodi, Hummelsheim, Dodd, Perlia, Campbell, Ohm, Holmes Spicer, Enslin, Faith, E. v. Hippel, Meissner, Pfalz, Terlinck). v. Hippel throws doubt upon the causal relationship of traumatism.

HILLEMANNS.—A. f. A., xxxi, 1895; xxxii, 1896. v. ARLT.—Ueber die Verletzungen des Auges, Wien, 1875. COOPER.—Injuries of the Eye, London, 1859. MOOREN.—Ophthalmiatrische Mittheilungen, 1873. SZILI.—A. f. A., xiii, 1884. GRANDCLÉMENT.—A. d'O., viii, 1888. MARKWORT.—A. f. A., xxi, 1890. ELIASBERG.—Westnik Ophth., vii. BRONNER.—T. O. S., ix, 1889. FUCHS.—Lehrbuch; A. f. O., xli, 4. SALZMANN.—In Praun. STOOD.—A. f. A., xliii, 1901. NIEDEN.—C. f. A., xv, 1891. HIRSCH.—Wochenschr. f. Therapie u. Hygiene des Auges, 1898. *PRAUN.—Die Verletzungen des Auges, Wiesbaden, 1899. HALTENHOFF.—Ann. d'Oc., cix, 1893. CRITCHETT.—Ann. d'Oc., lxxvii, 1877. SÉNUT.—Rec. d'O., 1886. HIRSCHBERG.—C. f. A., xvi, 1892. SCHMITZ.—Dissertation, Bonn, 1894. SCHIRMER.—Vossius' Sammlung, 1900. BERGMEISTER.—Z. f. Heilk., xxvi, 1905. HAASS.—C. f. A., xxix, 1905. MAYERHAUSEN.—Centralbl. f. d. med. Wissenschaft, 1885. BIETTI.—Ann. di Ott., xi, 1882. DODD.—Ophthalmoscope, 1904. v. REUSS.—C. f. A., xxv, 1901. BRONNER.—T. O. S., x, 1890. CSAPODI.—Ungar. med. Presse, 1896. HUMMELSHEIM.—Ophth. Klinik, 1904. PERLIA.—K. M. f. A., xliii, 1905. DE LIETO VOLLARO.—A. di Ott., 1905. POSEY.—Ophth. Rev., xxiv, 1905. ENSLIN.—Z. f. A., xv, 1906. FAITH.—Amer. Jl. of Ophth., 1906. *E. v. HIPPEL, PFALZ.—B. d. o. G., 1906. TERLINCK.—La Clinique ophth., 1906. CAMPBELL.—Med. Press, 1905. HOLMES SPICER.—Ophth. Rev., xxiv, 1905.

Burns of the conjunctiva and cornea, whether caused by hot fluids, hot match heads, etc., or by chemical caustics, injure or destroy the epithelium and subepithelial tissues to a greater or less degree. The chief deleterious effects are due to corneal necrosis and scarring, symblepharon, occlusion and distortion of the lacrymal puncta, and cicatricial displacements of the lids. Perforation of the globe may occur, with resultant anterior staphyloma or panophthalmitis. The sclerotic may be burnt. de Vincentiis has observed the formation of a large cyst reaching to the lower margin of the cornea in such a case.

Lime burns require special description. Lime or calcium oxide in contact with water becomes transformed into slaked lime or calcium hydrate. The chemical combination is accompanied with great evolution of heat (150° C). The worst results occur if the process takes place in the eye, but slaked lime or its mixture with sand, etc. (mortar) is also extremely caustic. Lime burns have been experimentally investigated by Gosselin, v. Gouvea, Gühmann, Stroschein, and others. v. Gouvea found that the epithelium was destroyed, the particles of lime penetrating the tissues and causing necrosis by the abstraction of water. The lime-containing scar tissue is intensely white and porcelain-like,

giving rise to an extremely dense cicatrix. According to Gühmann the lime exists in the tissues as calcium chloride, carbonate and phosphate, not as hydrate. According to Stroschein it combines with the proteid material to form an albuminate. Probably both conclusions are correct (*cf.* "Conjunctivitis petrificans," Vol. I, p. 92). According to Rosenthal and zur Nedden the lime acts upon the "mucoid" substances of the cornea. The treatment with sugar solution (Gosselin) is deleterious.

Instances of corneal injury by ammonia (Trousseau, Denig), nitronaphthalin and benzin (Silex), oil of mustard (Neustätter, Pick), etc., are reported.

GOSSELIN.—Arch. gén. de Med., 1855. v. GOUVEA.—A. f. A., i, 1869. GÜHMANN.—Inaug. Dissertation, Breslau, 1884. STROSCHEIN.—Zeitschrift f. ärztl. Landpraxis, 1892.—ANDREAE.—Die Verletzungen des Sehorganes mit Kalk, Leipzig, 1899. SCHMIDT-RIMPLER.—Berliner klin. Woch., 1900. TROUSSEAU.—Rev. gén. d'O., 1901. SILEX.—Z. f. A., v, 1901. NEUSTÄTTER, PICK.—C. f. A., xxv, 1901. ROSENTHAL.—Z. f. A., vii, 1902. DENIG.—Z. f. A., xi, 1904. STIEREN.—Ophth. Rec., 1904. PAGENSTECHER.—Ziegler's Beiträge, vii, Supplementband, 1905. GUILLERY.—A. f. A., xliv, 1902; lvi, 1907. ZUR NEDDEN.—A. f. O., lxiii, 1907.

The action of bright sunlight and of electric light upon the superficial parts of the eye (photophobia, ophthalmia electrica) is partly due to the heat rays, but more to the chemically active ultra-violet rays. The latter are alone effectual upon the lens and retina, the aqueous acting as a heat screen. Lightning acts chiefly through the ultra-violet rays, but in this case electrolytic and mechanical effects must be taken into consideration.

Bright light of the sun, as in viewing an eclipse with unprotected eyes, and of the electric light, especially the bright flash in short-circuiting (Roy, Hewetson, Panas, Uhthoff, Würdemann and Murray, Lundsgaard), causes a central positive and negative scotoma. In the earliest stage there may be a white spot at the macula, later giving place to pigmentation, with or without a central greyish area. Obstruction of a retinal artery (Batten) and thrombosis of a retinal vein (Walker) have been attributed to bright light. Neuro-retinitis has been recorded by Marquez, Menacho, and others ; optic atrophy by Ulbrich.

Experimental observations with bright light have been carried out by Czerny, Deutschmann, Widmark, Cassien, Aubaret, and others (*v.* Vol. III, p. 1022). Aubaret considers that when the natural reflex protective mechanism—contraction of the pupil, closure of the lids, etc., fails in its object each cell in the retina takes on its own defensive measures, which consist, as in unicellular organisms, in a withdrawal of its processes. If the injurious stimulus is extreme the retraction is permanent and the neuron chain is irremediably broken.

DUFOUR.—Bull. de la Soc. méd. de la Suisse Rom., 1879. DEUTSCHMANN.—A. f. O., xxviii, 3, 1882. SULZER.—K. M. f. A., xxi, 1883. DRUMMOND.—Med. Press, 1883. SWANZY.—Ophth. Rev., ii, 1883. FROST.—T. O. S., v, 1885. SCHNELLER.—A. f. O., xxxi, 1, 1885. BOCK.—C. f. A., xiv, 1890. MACKAY.—Ophth. Rev., xiii, 1894. BARRETT.—Ophth. Rev., xiv, 1895. DUANE.—A. of O., xxiv, 1895. ZIRM.—A. f. O., lx, 1905. TREACHER COLLINS.—R. L. O. H. Rep., xiv, 1896. ROY.—Amer. Jl. of O., 1897. HEWETSON.—Lancet, 1897. SIEGFRIED.—B. z. A., xxii, 1898. UHTHOFF.—Z. f A., ii, 1899. WÜRDEMANN AND MURRAY.—Ophth. Rec., 1899. ULBRICH.—C. f. A., xxiv, 1900. MENACHO.—Internat. Congress, Paris, 1900. BATTEN.—T. O. S., xxi, 1901. SNELL.—

Brit. Med. Jl., 1902. Panas.—A. d'O., xxii, 1902. Walker.—T. O. S., xxiii, 1903. Birch-Hirschfeld.—A. f. O., lviii, 1904. Beauvois.—Rec. d'O., 1906. Heckel.—Amer. Jl. of O., 1906. Lundsgaard.—K. M. f. A., xliv, 1906. Villard.—Ann. d'Oc., cxxxvi, 1906. Hess.—A. f. A., lvii, 1907. *Aubaret.—A. d'O., xxvii, 1907.

In ophthalmia electrica the lids swell, the conjunctiva is chemosed and injected, and there is severe pain as of a foreign body and lacrymation and photophobia.   Erosions of the cornea occur.   The iris is hyperæmic and the pupil contracted ; synechiæ have been described. The severer symptoms last 6—10 hours.   The condition has been reported by Nicolai, Prat, Maklakoff, and others ; it has been investigated experimentally by Ogneff.   The effects of light and ultra-violet rays upon the lens have already received attention (Vol. III, p. 1022).

Rockliffe.—Ophth. Rev., i, 1882. Emrys-Jones, Little.—Ophth. Rev., ii, 1883. Terrier.—A. d'O., viii, 1888. Martin.—Ann. d'Oc., c, 1888. Widmark.—Skand. Arch., i, 1889 ; iii, 1892 ; iv, 1893. Brose, Rivers.—A. of O., xxiii, 1894. Haab.—K. M. f. A., xxxv, 1897. Prat.—In Nagel's Jahresbericht, 1888. Maklakoff.—A. d'O., ix, 1889. Ogneff.—Pflüger's Archiv, lxiii, 1896. Oliver.—T. Am. O. S., 1897. Lundsgaard.— K. M. f. A., xliv, 1906. Alexander.—Deutsche med. Woch., 1899. Leitner.—In Nagel's Jahresbericht, 1897. Mettey.—Ann. d'Oc., cxxxi, 1903. Le Roux.—A. d'O., xxiv, 1904. Valois.—La Clinique opht., 1904.

The prominent feature in most cases of **injury by lightning** is the development of cataract (v. Vol. III, p. 1022), but cases are reported in which this was absent.   The cataract has been attributed to bright light (Himly), rupture of the capsule (Yvert), katalytic action (Leber, since rejected by him), contusion (Nagel and Schleicher), secondary to cyclitis (Vossius), concentration of salts in the aqueous (Peters), etc.   One thing is certain, the forces at work are complex, including mechanical shock of a severe character, heat, light, chemical activity of ultra-violet rays, electrolytic action, etc.   It is impossible in the present state of knowledge to define the relative value of these factors.   There is reason to eliminate heat as of much importance in affections of the deeper parts of the eye.   The cases of Meyerhöfer, Knies, and Silex, in which the cataract cleared up during observation, support the purely mechanical theory (concussion cataract).   There is no doubt that serious vascular engorgement follows lightning stroke (Kiribuchi), and that this effect upon the uveal tract, especially the ciliary body, may well lead to disturbance of the nutrition of the lens (v. Vol. III, p. 1008).

The conjunctiva often displays the characteristics of ophthalmia electrica.   The cornea may show temporary or permanent opacities (Vossius, Knies, Denig, Silex, Reinewald, Kiribuchi).   Buller saw hyphæma, Reinewald iritis, Vossius iridocyclitis, Uhle, Leber, and Vossius paralysis of accommodation, Vossius spasm of accommodation, Power, Uhle, Leber, and Meyerhöfer mydriasis, Pagenstecher and Laker miosis.   Rupture of the choroid was noted by Reich and Buller, choroiditis by Reinewald.   Vitreous opacities are common.   Hyperæsthesia of the retina is reported by v. Graefe and Purtscher, retinal hæmorrhages by Downar, Reich, Laker and Yarr.   The optic nerve showed anæmia in the case of Uhle, hyperæmia in those of Saemisch and Reich, neuritis in those of Pagenstecher, Brière, Laker, and Vossius, partial or total atrophy in those of Leber, Pagenstecher, Vossius, Rohmer, Buller, Treacher Collins, and Kiribuchi (experimental).

The frequent cases of cataract (*v.* Bibliography) show individual peculiarities. The cataracts are usually bilateral, develop gradually during weeks or months, are generally partial, but often total. Spontaneous resolution occurs rarely (Knies, Silex). Partial opacities are punctate or linear, or retiform (Schleicher). Posterior cortical cataract is described by Leber, Pagenstecher, Knies, Buller, and points to defective nutrition from disease of the ciliary body.

There is some analogy between lightning cataract and glass-blowers cataract (*v.* Vol. III, p. 1021 ; Robinson, Snell, Thompson, Cramer.)

Paresis of external ocular muscles is recorded by Saemisch, Pagenstecher, Vossius, and Buller ; diplopia by Uhle ; ptosis by Saemisch, Power, Pagenstecher, Knies, Uhle, Schleicher.

Without cataract.—SAEMISCH.—K. M. f. A., ii, 1864. POWER.—St. George's Hosp. Rep., v, 1871. V. GRAEFE.—K. M. f. A., iii, 1865. REICH.—K. M. f. A., xvi, 1878. UHLE. —K. M. f. A., xxiv, 1886. PURTSCHER.—A. f. O., xxix, 4, 1883. BRIÈRE.—Gaz. des Hôp., 1876. DENIG.—Münch. med. Woch., 1895. ROHMER.—A. d'O., xxv, 1895. WENDRINER.— Dissertation, Berlin, 1905.
With cataract.—SERVAIS.—Ann. d'Oc., lii. DOWNAR.—C. f. A., ii, 1878. GALEZOWSKI. —Rec. d'O., 1881. LEBER.—A. of O., xxviii, 3, 1882 (Bibliography). PAGENSTECHER.— A. f. A., xiii, 1884. LAKER.—A. f. A., xiv, 1885. YARR.—T. O. S., xxi, 1901. VOSSIUS.— Berliner klin. Woch., 1886; B. z. A., iv, 1892. KNIES.—A. f. O., xxxii, 3, 1886. HESS.— B. d. o. G., 1888. MEYERHÖFER.—K. M. f. A., xxiv, 1886. SILEX.—A. f. A., xviii, 1888. BULLER.—A. f. A., xxi, 1890. SCHLEICHER.—Inaug. Dissert., Tübingen, 1888. REINEWALD. —Inaug. Dissert., Giessen, 1895. TREACHER COLLINS.—T. O. S., xxiii, 1903. KIRIBUCHI.— A. f. O., I, 1, 1900. JUNIUS.—Ophth. Klinik., 1906. ROBINSON.—Brit. Med. Jl., 1903, 1907. SNELL, TATHAM THOMPSON.—Brit. Med. Jl., 1907. CRAMER.—K. M. f. A., xlv, 1907. BRIXA.—K. M. f. A., xxxviii, 1900. PREINDESBERGER.—Wiener klin. Woch., 1900. GINSBURG.—In Nagel's Jahresbericht, 1906. GONIN.—Ann. d'Oc., cxxxi, 1904.

II. INJURIES BY BLUNT OBJECTS.

The eye is protected to a considerable extent by the orbital bones, lids, etc., from direct injury. The anatomical disposition of parts affords least protection at the lower and outer side of the orbit. Severe contusions by blunt objects may injure the superficial and exposed parts of the globe, with or without causing rupture of the walls ; the deeper tissues may be contused indirectly, displaced, or ruptured, also with or without rupture of the sclerotic or cornea. The resultant injuries may be caused by the direct action of the blow or by the indirect distribution of the forces, the mechanism of which is often difficult to ascertain.

CORNEA.

Contusion of the cornea may result in simple abrasion or circumscribed opacification. Radial ruptures of Descemet's membrane may occur. The sensitiveness of the cornea is often depressed or abolished, and diminution of intra-ocular pressure may ensue without other apparent injury to the secretory and excretory mechanisms of the eye. More severe injury leads to rupture of the globe, the sclerotic almost invariably giving way, with or without implication of the cornea.

Contusion of the cornea may be followed by the development of filamentary keratitis (Vol. I, p. 183). Of twenty cases of this condition

eleven were due to herpes corneæ, eight to slight injury, and one to unknown cause (Wagenmann). Striate opacity may also occur (Vol. I, p. 179). Allied to these conditions is the formation of vesicles upon the surface of the cornea, with or without extravasation of blood into the blebs or substantia propria. Such cases are to be distinguished from those of blood staining of the cornea (v. Vol. I, p. 249). According to Zander and Geissler vesicular keratitis occurs especially after burns. Kleinschmidt noted it five weeks after injury with gunpowder. Vesicular keratitis with extravasation of blood has been reported by Schmidt-Rimpler, Mayerhausen, Rumschewitch, Dimmer. Rampoldi, Galezowski, Kauffmann, Scheffels and others record cases of blood infiltration after injury. Stellwag considers this most likely to happen in eyes with degenerate vessel walls, especially in elderly people with cyclitis. Czermak attributes the extravasation to rupture of Schlemm's canal, and some cases are undoubtedly due to rupture of Descemet's membrane in the presence of a hyphæma.

WAGENMANN.—B. d. o. G., 1892. KLEINSCHMIDT.—Inaug. Dissert., Bonn, 1876. SCHMIDT-RIMPLER.—K. M. f. A., xiii, 1875. MAYERHAUSEN.—C. f. A., vii, 1883. RUMSCHE-WITSCH.—C. f. A., viii, 1884. DIMMER.—K. M. f. A., xxiii, 1885. RAMPOLDI.—Ann. di Ott., xvii, 1888. KAUFFMANN.—Ophth. Klinik, 1901. SCHEFFELS.—Z. f. A., v, 1901. GALEZOWSKI.—Rec. d'O., 1887. STELLWAG.—Lehrbuch. LANDMANN.—A. f. O., xxviii, 2, 1882. CZERMAK.—K. M. f. A., xxvii, 1889. HUWALD.—A. f. O., lix, 1904.

Rupture of the cornea is rare. It occurs more frequently in young patients than in adults (L. Müller); of all recorded cases only four

FIG. 774.—BIRTH INJURY.
Thomson, T. O. S., xxii. Showing corneal opacities.

patients were over twenty years of age. Cases are reported by Zander and Geissler, Becker, Chisholm, Hjort, Fuchs, Müller, and others. According to Müller half the cases are due to direct, half to indirect rupture, in marked opposition to scleral rupture in which direct rupture is extraordinarily rare. Unlike scleral rupture, too, there is no special

predilection of site; any portion of the cornea may give way. In some cases the site has been weakened by previous disease, *e.g.* ulceration (R. L. O. H. Museum, I, B, 10), cataract cicatrix (R. L. O. H. Museum, I, B, 19), etc. (Mackenzie, Müller, and others). Naturally the part exposed in the palpebral aperture suffers most frequently, and horizontal ruptures extending into the limbus are relatively common. Linear, radiate, and curved ruptures are described.

Simultaneous involvement of deeper parts of the eye is common — iridodialysis, etc., prolapse of iris, irideremia, hyphæma, traumatic cataract, vitreous hæmorrhage. The retina and choroid usually escape; sympathetic ophthalmia has not been observed as a sequel, but infection and panophthalmitis occur.

FIG. 775.—BIRTH INJURY. Thomson and Buchanan, T. O. S., xxiii. Diagram of posterior surface of cornea, showing gaps in Descemet's membrane, which is stained with blood.

ZANDER AND GEISSLER.—Ueber die Verletzungen des Auges, Leipzig, 1864. BECKER.—Atlas, Tafel xxiv. TALKO.—K. M. f. A., xxix, 1891. HJORT.— K. M. f. A., xiv, 1876. FUCHS.—Lehrbuch. *L. MÜLLER.—Ueber Ruptur der Corneoscleralkapsel durch stumpfe Gewalt, Wien, 1895. PRAUN.—Die Verletzungen des Auges, Wiesbaden, 1899.

Injury of the cornea at birth forms an interesting group of indirect
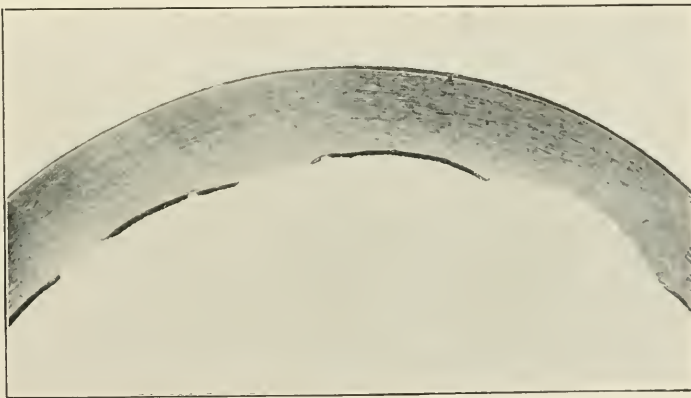
FIG. 776.—BIRTH INJURY. × 15.
From the same specimen. Showing three gaps in Descemet's membrane, two of which involve also the posterior corneal lamellæ.

injury. Cases have been described by de Wecker (1896), Truc (1898), and have been carefully investigated by Thomson and Buchanan (1903). In all cases labour was difficult and forceps were used. Three varieties of change in the cornea have been noticed: (1) A diffuse opacity

which is temporary; it is due to œdema. (2) A diffuse opacity, indeterminate in position, which is permanent; it is due to œdema, probably with consecutive inflammatory changes, and rupture of
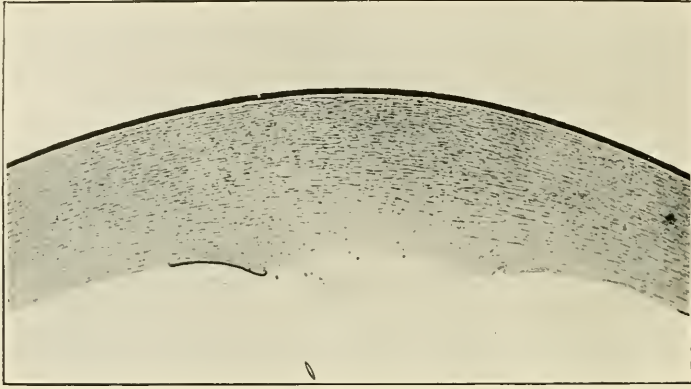
FIG. 777.—BIRTH INJURY. × 18.
Thomson and Buchanan, T. O. S., xxiii. Showing rupture of the posterior layers of the cornea.
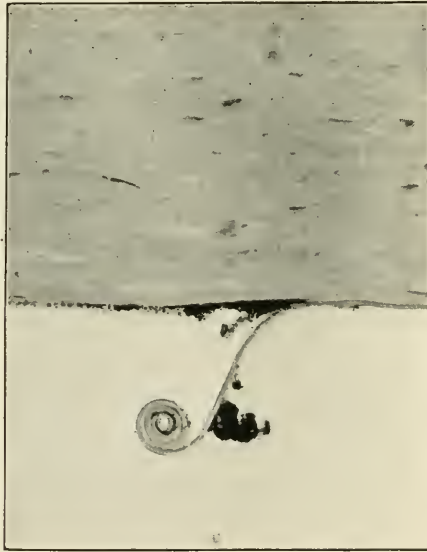
FIG. 778.—BIRTH INJURY.
Thomson and Buchanan, T. O. S., xxiii. Posterior part of cornea under high power, showing rupture of Descemet's membrane, with heaping up of pigment.

Descemet's membrane. (3) An opacity which takes a linear form and is permanent; it is due to rupture of Descemet's membrane and

sometimes of posterior corneal lamellæ. The cases of de Wecker, Truc, and some of those of Thomson and Buchanan belong to the last category. It is probable that in some of the cases the injury is due to direct pressure of the blade of the forceps on the cornea. E. v. Hippel has noticed rupture of Descemet's membrane without laceration of the corneal substance. He considers that it may be due to sudden increase of intra-ocular pressure during birth. Traumatic keratitis was seen by Thomson and Buchanan in eight cases, involving twelve eyes. The opportunity of observing so many cases is owing to the prevalence of rickets and contracted pelves in Glasgow.

Pfuhl.—Charité Annalen, 1883. Nagel.—Arch. f. Gynäk., xxxix, 1891. Dittrich.—Wiener klin. Woch., 1892. Noyes.—T. Am. O. S., 1895. de Wecker.—Ann. d'Oc., cxvi, 1896. Dujardin.—Jl. de Méd. et Chir. pratiques, 1897. Truc.—Ann. d'Oc., cxix, 1898. Servel.—Thèse de Lyon, 1901. Thomson.—T. O. S., xxii, 1902. Cargill.—T. O. S., xxii, 1902. *Thomson and Buchanan.—T. O. S., xxiii, 1903. Sidler-Huguenin.—Korrespondenzbl. f. Schweizer Aerzte, 1903. Fejér.—C. f. A., xxviii, 1904. *Wolff.—Hirschberg's Festschrift, Leipzig, 1905. Stephenson.—Ophthalmoscope, 1905. Peters.—A. f. A., lvi, 1906.

Sclerotic.

Rupture of the sclerotic is either direct or indirect. Direct rupture is even rarer than direct rupture of the cornea. It occurs at the spot injured, and varies according to the nature of the injury. A case is described by L. Müller.

Typical indirect rupture of the sclerotic is common. Sachs collected 114 cases from the literature up to 1889, but this gives no criterion of its frequency. L. Müller described 45 cases from Fuchs's clinic alone. Early records date from 1583 (Bartisch), with subconjunctival dislocation of the lens; 1799 (Demours); 1813, from injury with a cow's horn (Beer); 1835, with subconjunctival dislocation of the lens (Middlemore); 1839 (Mackenzie); 1849 (Rivaud-Landrau); 1850 (Barrier); 1852 (Sichel); 1854 (v. Graefe, Jaeger). Of 97 of Sachs's cases 23 were from blows with a cow's horn, 19 from blow or fall against a table, box, bed, etc., 14 from the end of a stick, tube, etc., 8 from blows with the fist, 6 from the finger, and so on. One third of L. Müller's cases were due to blows with an animal's horn (see especially Ernst Schmidt, Goldberg, Bertram, Boerner, Purtscher). The older authors laid stress upon the advanced age of the patients. Briolat found 1 case at 17, 3 between 30 and 40, 4 between 40 and 50, 6 between 50 and 60, 7 between 60 and 70. L. Müller found 18 before 40 and 26 after. Berlin attributed the cause to the hardness of the lens; the loss of resilience of the sclerotic is a more important factor (Falchi). Scleral ruptures in youth are recorded by Nuel (2 years), Kayser (15 years), Cooper (17 years), Trelat and Massie, Zander and Geissler, and others.

The rupture is very constant in its relationship to the limbus. It is almost always concentric with it and a few millimetres behind it— 2—5 mm. (v. Arlt), 2—4 mm. (Becker), 2 mm. (Briolat), at most 3 mm. (Nuel). Rare cases in other situations have been recorded—in the equator (Nuel, Weeks), meridional (Fano, Schröter), in the posterior

hemisphere (Chisholm, Bowman). The rupture is generally 10—12 mm. long or more, minimum 3 mm.; often one third to one half the limbal circumference is involved, the true limits being only discovered by anatomical investigation. The tension is, of course, very low. The site is usually above the horizontal meridian—36 above, 20 in, 21 in and up, 11 out and up, 8 out, 2 down and in, 2 down and out (Sachs). Rupture downwards has been recorded by Carré, Alt, Falchi, Rau, Schäfer, Schröter, Wintersteiner, E. Schmidt. L. Müller records the site of incidence of the blow in 29 cases—5 up and out or in and up and out, 3 in and down, 2 down, 2 down and out. In 17 of the 29 cases the middle of the rupture was 90° from the site of incidence of the blow, in 3 less than 90°, in 5 greater. In 20 cases the foreign body penetrated 11 times between the orbital margin and the eye—6 times above, 2 below, 1 in, 1 in and above and out. In no case was the sclera struck down and out, nor
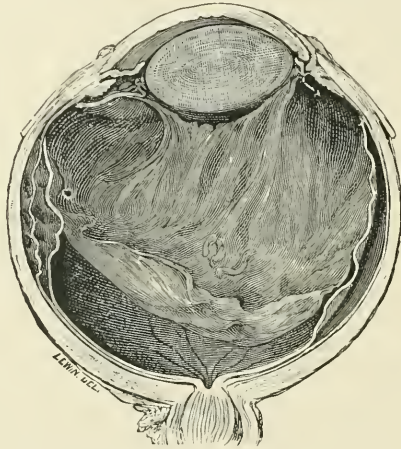
FIG. 779.—RUPTURE OF GLOBE. × 2½.
R. L. O. H. Museum, Series I, Sub-series B, No. 13. Rupture of sclerotic, with prolapse of iris and displacement of lens and vitreous.

did the foreign body penetrate in this direction between the orbital margin and the eye; in no case is the cornea given as the site of incidence.

The force of the injury is generally so great that there is severe injury to other parts of the eye. Ecchymosis of the lids, subconjunctival hæmorrhage, abrasion of the cornea, hyphæma, vitreous hæmorrhage (with early resolution—Fano) are almost constant concomitants. The lips of the wound are everted in recent rupture, inverted after cicatrisation. The iris may be intact and *in situ*, even with subconjunctival luxation of the lens. Frequently there is iridodialysis at the site of rupture. Radial tears, retroversion, prolapse, aniridia, occur. The ciliary body is often implicated. The lens is almost always displaced—lateral into the wound, out of the wound (subconjunctival) or eye (Cooper, Mules, Szili, Fuchs, and others), into the vitreous, etc. The capsule is usually ruptured. The vitreous is

generally prolapsed. The retina and choroid often escape injury, though all the contents of the globe may be expelled.

In 14 cases Landesberg found subluxation of the lens twice, retinal hæmorrhage twice, vitreous hæmorrhage twice, rupture of the lens capsule once, choroidal rupture once. Sachs notes a round pupil in two cases in the literature in the presence of luxatio lentis; complete irideremia occurred in 17 of 83 cases, apparent coloboma in 39. Only in 3 cases was the lens in its normal position. Subconjunctival dislocation occurred in 77 out of 123 cases, whilst the lens was expelled from the eye in 30, though the latter result is the commonest, the cases not being published. Similar statistics are given by L. Müller, who also records the second published case of dislocation of the lens into Tenon's capsule.

Phthisis bulbi usually follows if the eye is retained, with or without suppuration and panophthalmitis. In the absence of infection a depressed scar is formed or scleral extasia follows from stretching of the cicatrical tissue (cf. Landesberg). Schrag observed stretching of the posterior pole with development of high myopia. Secondary glaucoma often occurs. Vitreous opacities are a frequent sequel, with new-formed fibrous tissue (" retinitis proliferans "). Retinal hæmorrhages, pigmented and atrophic spots in the choroid, etc., are seen. In many cases sympathetic ophthalmia (q. v.) has developed (Bartisch (1583), Barrier (1850), Cooper (1860), Lawson (1865), Schröter (1866), Schirmer, L. Müller, Knapp). Panophthalmitis is relatively uncommon, owing to the indirect nature of the injury.

Rare complications are dislocation of the globe and tetanus (L. Müller). Pigmentation of the conjunctiva, generally from iris pigment (often with irideremia), less often from blood pigment, is recorded (Wintersteiner, Caspar, Kiranow, Hirsch, Praun).

The mechanism of scleral rupture has been the subject of much conjecture. Stellwag (1855) considered that the greater corneal curvature opposed less resistance to the stress of the blow than the neighbouring sclera; the cornea becomes indented, the sclerotic is stretched and gives way in a meridional direction. Zander and Geissler (1864) thought that the blow forced the globe against the upper margin of the orbit. Manz (1865) held that a blow directed from the outer or outer and lower side, the usual directions, forced the eye up or up or in; it is then supported on every side except up and in, and this part of the sclerotic therefore ruptures. Lawson (1866) attributed the site to the diminishing thickness of the sclera from behind forwards, the posterior parts being further supported by the extrinsic muscles and contents of the orbit. Schröter (1866) supported Manz's views and held that the site of rupture is always opposite the site of incidence of the blow. Berlin (1873) discussed the question when treating of commotio retinæ (q. v.). v. Arlt (1874) pointed out that the globe is compressed in the direction of incidence of the force: since the contents are incompressible the walls must be stretched in the direction at right angles, and the tendency to stretching will be greatest in a great circle of the bulbar sphere; this part is supported, however, by the orbital contents, so that the greatest stress is felt in

the unprotected portion farther forwards, which therefore ruptures. Massie (1875) and Briolat (1879) content themselves with the explanation that the globe gives way at the weakest spot.   Kern's explanation is similar to Manz's, importance being attached to the support of the globe under the sudden rise of intra-ocular pressure at the point which receives the force of the blow.   Hughes (1887) attempts to prove mathematically that the rupture must take place where the " Umbiegungsring " meets the corneal margin—the " Umbiegungsring " being the line which delimits the part impressed by the foreign body from the neighbouring compensatory protruding part of the sclerotic. Sachs (1889) considers that the aqueous is forced under great pressure into its normal channels of excretion, finding in the canal of Schlemm a punctum minoris resistentiæ.   L. Müller (1895) holds that the direction of the blow is seldom from down and out as generally accepted, but rather up and in ; there is also no closure of the lids and rolling upwards as v. Arlt, Senfft, and others state.   The analogy with fracture of the skull is on many grounds fallacious.   Blows directed from before backwards are little liable to cause scleral rupture on account of the elastic support from the orbital contents ; such blows usually cause rupture of the choroid (q. v.).   Compression of the bulbar contents forces the globe to approximate as nearly as possible a perfect sphere, as shown by v. Helmoltz ; hence the circumcorneal groove is abolished, and as soon as this occurs rupture takes place.   The anatomical structure of the sclerotic is obviously a very important factor.   The thinnest part is just behind the insertion of the muscles, yet rupture never occurs here owing to external support.   Neither does it occur at the true corneo-scleral margin.   In the intermediate zone the ligamentum pectinatum, the canal of Schlemm, and the presence of the anterior perforating vessels lead to weakening.   Experiments on the breaking strain of meridional strips of cornea and sclerotic show that rupture under these conditions occurs, as might be expected, at the thinnest spot (Stoewer) ; they have little bearing on the subject.

It has been pointed out that the rupture is most frequently above the horizontal meridian of the cornea.   Manolescu (1885) attributes this to to the support of the lower lid.   L. Müller is convinced that no closure of the lids occurs, and attributes importance to pressure of the trochlea of the superior oblique at the site of extreme tension of the globe.

There is no doubt that the sclerotic tears from within outwards. The inner wound is anterior to the outer ; the walls rupture in the order sclerotic, episclera, and conjunctiva.

Experimental observations have been made by Follin (1853), v. Seidlitz (1873), Berlin (1873), and L. Müller (1895) on the cadaver ; the results throw little light on the subject.   Roquette (1892) showed that rupture of the human eye occurs under a pressure of 8—9 kilos, of the ox eye under 25—27 kilos.

*L. Müller.—Ueber Ruptur der Korneoscleralkapsel durch stumpfe Gewalt, Leipzig u. Wien, 1895.   *Sachs.—A. f. A., xx, 1889.   Demours.—Traité des Maladies des Yeux, 1818.   Beer.—Lehrbuch der Augenerkrankungen, Wien, 1813.   Ernst Schmidt.— Inaug. Dissert., Giessen, 1895.   Goldberg.—Dissertation, Freiburg, 1898.   Bertram.—

Dissertation, Göttingen, 1901. BOERNER.—Dissertation, Halle, 1902. BERLIN.—K. M. f. A., xi, 1873. BRIOLAT.—Thèse de Paris, 1879. FALCHI.—Ann. di Ott., xiv, 1885. v. ARLT.— K. M. f. A., xii, 1874. BECKER.—K. M. f. A., xvi, 1878. NUEL.—Ann. d'Oc., xcix, 1888. COOPER.—On Wounds and Injuries of the Eye, London, 1859. MULES.—T. O. S., vii, 1887. SZILI.—A. f. A., xiii, 1884. FUCHS.—Lehrbuch; Wiener klin. Woch., 1905. LANDESBERG. —A. f. A., xvii, 1888. SCHRAG.—Dissertation, Berlin, 1870. SCHIRMER.—A. f. O., xxxviii, 4, 1892. KNAPP.—T. Am. O. S., 1893. WINTERSTEINER.—Wiener klin. Woch., 1893. CASPAR.—K. M. f. A., xxxi, 1893. KIRANOW.—B. z. A., xxiv, 1898. HIRSCH.—B. z. A., xxvi, 1898. *PRAUN.—Die Verletzungen des Auges, Wiesbaden, 1899. STELLWAG.—Die Ophthalmologie vom naturw. Standpunkt, ii, 1855. MANZ.—K. M. f. A., iii, 1865. LAWSON, SCHRÖTER.—K. M. f. A., iv, 1866. MASSIE.—Thèse de Paris, 1875. KERN.—Deutsche militärärztl. Zeitschrift, xv. *HUGHES.—A. f. O., xxxiii, 3, 1887. SENFFT.—Dissertation, Kiel, 1896. STOEWER.—A. f. A., xxiv, 1892. MANOLESCU.—A. d'O., v, 1885. FOLLIN.— Arch. de Méd., 1853. v. SEIDLITZ.—Dissertation, Kiel, 1873. ROQUETTE.—Thèse de Lyon, 1892. *PURTSCHER.—Hirschberg's Festschrift, Leipzig, 1905.

Posterior rupture of the sclerotic.—Rupture of the posterior parts of the sclerotic by direct injury, *e. g.* bullet wounds, is common. Indirect injury may cause rupture, concentric with the disc (Mules, Chisholm). In Mules's case a piece of brick fell upon the eye, which was directed upwards. The papilla, together with an irregular zone of sclera, was forced outwards.

MULES.—T. O. S., vii, 1887. CHISHOLM.—A. of O., xi, 1882.

Partial rupture of the sclerotic.—It has already been pointed out that complete rupture takes place from within outwards. Occasionally the outer layers of the sclerotic or the episclera, as well as the conjunctiva, escape. Such partial ruptures, leading to ectasia, have been reported by v. Arlt and L. Müller.

Rupture of the canal of Schlemm occurs with direct injury to the limbus. It was proved experimentally by Berlin, and cases have been collected by Czermak. It is really a partial scleral rupture, only the inner lamellæ, involving the canal of Schlemm, being implicated. The anterior chamber fills with blood, and iridodialysis may also be present (Schäfer, Williams [?], Panas).

BERLIN.—K. M. f. A., xi, 1873. CZERMAK.—K. M. f. A., xxvii, 1889. SCHÄFER.— A. f. O., xxix, 1, 1883. WILLIAMS.—K. M. f. A., xxvii, 1889. PANAS.—Maladies des Yeux, Paris, 1894.

IRIS.

Hyphæma.—Injury to the eye with a blunt object—wood, iron, fist, &c.—often results in hyphæma. Minute hæmorrhages into the iris tissue may occur. Rupture of larger iris vessels, often associated with iridodialysis, irideremia, or radial tears in the membrane, or of the canal of Schlemm (*vide supra*) cause more extensive extravasation of blood, which sinks to the lowest part of the anterior chamber. Deeper seated injuries are often concomitant—vitreous hæmorrhage, rupture of the choroid, detachment of the retina, etc. The blood is usually absorbed in 3—8 days, but recurrent hæmorrhages occur (Cooper, Bowman, Praun), usually in diseased or disorganised eyes. Thus, recurrent spontaneous or very readily induced hæmorrhages are frequent in eyes which have suffered from long-standing iridocyclitis,

absolute glaucoma, arterio-sclerosis, etc. Extravasation of blood into Petit's canal is illustrated in Jaeger's 'Atlas.' Hyphæma is not an infrequent birth injury (Lomer, Wintersteiner, Leopold, Sidler-Huguenin, Thomson and Buchanan, Bylsma, Klauer, Volkmann, Hochstetter, Wolff).

COOPER.—On Wounds and Injuries of the Eye, London, 1859. WEBER.—A. f. O., vii, 1860. BOWMAN.—In Zander and Geissler. PRAUN.—Die Verletzungen des Auges, Wiesbaden, 1899. LOMER.—Z. f. Geburtshilfe, x, 1884. WINTERSTEINER.—Z. f. A., ii, 1899. LEOPOLD.—C. f. Gynäk., 1902. SIDLER-HUGUENIN.—Correspondenzbl. f. Schweizer Aerzte, 1903. THOMSON AND BUCHANAN.—T. O. S., xxiii, 1903. BYLSMA.—Münchener med. Woch., 1901. KLAUER, VOLKMANN, HOCHSTETTER.—Charité Annalen, 1893–98. WOLFF.—Hirschberg's Festschrift, Leipzig, 1905.

Iridodialysis.—The weakest part of the iris is the peripheral zone close to the insertion into the ciliary body, as is shown by the ease

FIG. 780.—IRIDOIALYSIS.
Nettleship, Diseases of the Eye. Multiple iridodialysis following a blow.

with which it is torn in this situation in iridectomy for acute glaucoma. It has long been known that partial rupture may occur here as the result of non-perforating injuries (Ried, 1847). v. Arlt considered that flattening of the cornea dilates the zone of attachment of the iris. Förster attributed the principal *rôle* to the aqueous, which is forced backwards bodily; the iris is suddenly pressed back, with dilatation of the pupil, rupture of the sphincter, or iridodialysis, partial or complete (aniridia), according to the force and incidence of the blow. Partial or complete dislocation of the lens may follow from the same cause. L. Müller found that dialysis is especially frequent when the blow falls upon the limbus, owing to stretching of the iris attachment in this neighbourhood. The blood of the associated hyphæma may be derived from ruptured iris vessels, from the circulus arteriosus iridis major (lying in the ciliary body), or from the canal of Schlemm. Wintersteiner thinks that it is usually from the last source.

The dialysis may be of any size and in any situation, but is generally above. Occasionally multiple dialyses occur, though Fuchs states that he has never seen multiple ruptures as the result of injury with a blunt object. Wintersteiner explains them as due to one or more small prolapses, completely covered with conjunctiva. In other cases one dialysis may be due immediately to the blow, another to dislocation of the lens (L. Müller). Iridodialysis may occur at birth with forceps delivery (Bylsma); also partial aniridia (E. v. Hippel).

The activity of the sphincter causes the iris to be pulled towards the centre of the pupil at the site of dialysis, so that the pupil ceases to be round. The detached part of iris may overlap the uninjured part, or may cover the pupil. The detached portion never heals into its normal position, even under atropin (*cf.* Charles). In the course of years further detachment may occur, even to complete irideremia. In other cases the gap may become filled in with fibrous tissue (R. L. O. H. Museum, I, A, 23). Doremaal records the complete absorption of

separated particles of iris tissue. In the absence of other complications neither glaucoma nor phthisis bulbi result, nor is there much inflammatory reaction. Anatomical examinations have been made by Alt, Schiess, Treitel, Schäfer, Wintersteiner, L. Müller.

FÖRSTER.—B. d. o. G., 1887. L. MÜLLER.—Ueber Ruptur der Corneoskleralkapsel durch stumpfe Verletzung, Wien, 1895. BYLSMA.—Woch. f. Therapie u. Hyg. des Auges, 1901. E. v. HIPPEL.—A. f. O., lii, 1901. WINTERSTEINER.—A. f. O., xl, 2, 1894. DORE-
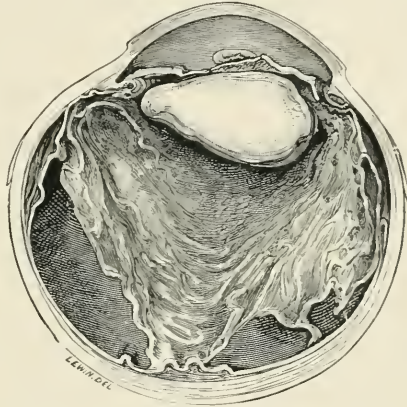
FIG. 781.—IRIDODIALYSIS. × 2½.
R. L. O. H. Museum, Series I, Sub-series A, No. 31. Prolongation outwards of the angle of the anterior chamber on one side and detachment of the iris from the ciliary body on the other.

MAAL.—Geneeskundige Courant, 1889. BALLABAN.—C. f. A., xxiv, 1900. CRETSCHMAR.—Z. f. A., v, 1901. SWEET.—Ophth. Rec., 1901. TEICH.—A. f. A., lii, 1905. CHARLES.—Amer. Jl. of O., 1906.

Traumatic aniridia or irideremia.—This is, strictly speaking, total iridodialysis, but anatomical investigation has shown that many of the cases observed clinically are not really complete (Wintersteiner). The condition is very rare without rupture of the globe. In these rare cases the mechanism is the same as in partial iridodialysis (q. v.) ; in the others the tearing away of the iris is facilitated by the outflow of aqueous and the dislocation of the lens (L. Müller). After the absorption of hyphæma the appearances are similar to those of congenital aniridia, though the rolled-up iris may be visible as a black ball in the anterior chamber ; it may have disappeared entirely, lying probably behind the scleral margin, for it is never completely absorbed (Delacroix). The lens is often dislocated ; in some such cases the eye looks conical owing to the flattening of the circumcorneal groove (Dixon, Vose Solomon, Praun). Vitreous hæmorrhage is common, and the dark red blood can be seen unusually well by oblique illumination. The ciliary processes may be clearly visible, or it may be impossible to distinguish them (Haltenhoff, Samelson, Lyder Borthen, Praun). In the latter case it does not follow that the iris has been retroflexed, still less that the processes have been torn away or were

congenitally absent (Samelson). Most probably they are masked by
new-formed connective tissue, which may drag them forwards and
cause them to adhere even to the back of the periphery of the cornea
(Wintersteiner). More commonly they are doubtless hidden behind a
mass of fibrous tissue, to the back of which they are adherent. In rare
cases it can be seen clinically that the dialysis is not completely round
the circle (Schäfer, Pagenstecher, Briolat, Schröter, Samelson, Praun).
When rupture of the globe has also occurred the iris is generally
carried out of the eye with the lens; occasionally it is incarcerated in
the wound, heals under the conjunctiva, or remains in the anterior
chamber.

Some vision may be recovered even in cases in which the lens is
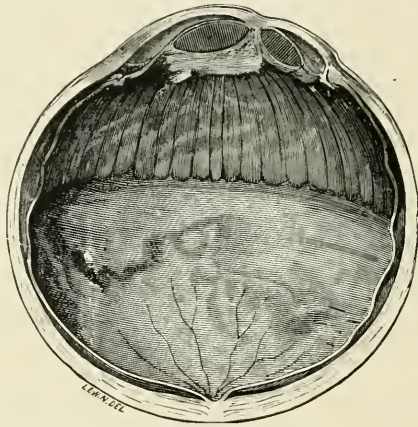
FIG. 782.—TRAUMATIC ANIRIDIA.   × 2½.
R. L. O. H. Museum, Series I, Sub-series C, No. 4.   Complete absence of iris,
adhesion of lens capsule to cornea, and cupping of optic disc.

dislocated (Lyder Borthen, Praun, Nunnely, Haltenhoff, Schaligin).
Cases of traumatic aniridia without displacement of the lens have been
recorded by Chisholm, Folker, Robertson, Hjort, Lange, Gayet,
Hirschberg, Carré, and others. There is the same tendency to
secondary glaucoma in traumatic as in congenital aniridia (q. v.)

*WINTERSTEINER.—A. f. O., xl, 2, 1894. L. MÜLLER.—Ueber Ruptur der Corneoscleral-
kapsel durch stumpfe Verletzung., Wien, 1895. DIXON.—In Zander and Geissler, Die Verlet-
zungen des Auges, Leipzig, 1869. PRAUN.—Die Verletzungen des Auges, Wiesbaden,
1899. HALTENHOFF.—Ann. d'Oc., lxxvi. SAMELSON.—Brit. Med. Jl., 1872; 1880. SAMEL-
SOHN.—C. f. A., iv, 1880. LYDER BORTHEN.—K. M. f. A., xxi, 1883. SCHÄFER.—A. f. O.,
xxix, 1, 1883. NUNNELY.—Brit. Med. Jl., 1870. SCHALIGIN.—Med. Bote, 1872. CHISHOLM,
FOLKER, &c.—See AHLSTRÖM, B. z. A., xvi. GOTTI—Boll. d'Oc., xiv, 1892. MACKINLAY.—
T. O. S., x, 1890. ARNOLD LAWSON.—T. O. S., xxiv, 1904. SHAW.—T. O. S., xxvii, 1907.
V. BOGUSZ.—Wiener med. Woch., 1900. ROWAN.—Ophth. Rev., xix, 1900. FEJÉR.—
A. f. A., xlviii, 1903. GEISSLER.—Wiener med. Woch., 1903. DE SCHWEINITZ.—Ophth.
Rec., 1905.

Retroflexion of the iris was first described by v. Ammon; it must
not be mistaken for incarceration of the iris in a rupture. Early cases
are recorded by J. A. Schmidt, Samelson, Taylor. The whole iris may

be retroflexed, so that a mere rim is seen (Parisotti). More commonly only a sector is affected, so that the appearance of a coloboma is given. The ciliary processes are not seen, being covered by the iris. The edge of the lens, usually dislocated, is visible. Traumatic mydriasis is frequent, and atropin and eserin are without effect. The accident may happen during the performance of iridectomy, etc. (de Wecker, Praun, Passauer). Partial or complete rupture of the zonule of Zinn probably facilitates retroflexion of the iris, and may be *sine quâ non.* Neither inflammatory nor glaucomatous sequelæ generally occur.

Förster ascribes retroflexion of the iris to a similar mechanism to that inducing iridodialysis (q. v.). It is difficult to explain on these grounds when rupture of the globe has also occurred, since the action of the aqueous can only be potent before the globe bursts. In those cases in which it occurs during the performance of an intra-ocular operation probably rupture of the zonule of Zinn precedes it, possibly inducing a negative pressure by separating the lens from the vitreous. Praun's case indicates that such a negative pressure may occur and act in this manner through loss of vitreous.

v. AMMON.—A. f. O., i, 2, 1855. J. A. SCHMIDT.—In Zander and Geissler. SAMELSON.—Brit. Med. Jl., 1872. TAYLOR.—Lancet, 1873. PARISOTTI.—Boll. dell'Acad. med. di Roma, xviii. DE WECKER.—In G.-S., iv, 1876. PASSAUER.—A. f. O., xix, 2, 1873. PRAUN.—Die Verletzungen des Auges, Wiesbaden, 1899. FÖRSTER.—B. d. o. G., 1887. HUBBEL.—Ophth. Rec., 1901.

Rupture of the sphincter pupillæ.—Cooper first drew attention to this injury, reporting four cases. Franke (1886) collected the previously recorded cases. The injury is not uncommon. Usually the ruptures are multiple, 2—8, extending 1 or 2 mm. from the pupillary margin of the iris, and involving the whole thickness. The pupil is generally moderately dilated, rarely fully, very rarely normal (de Wecker). There is usually a small hyphæma. Iridodialysis is rare as a complication (Josenhaus, Yvert), larger tears to the ciliary border not infrequent. Dislocation of the lens, rupture of the lens capsule (Schirmer) ; vitreous hæmorrhage, detachment of the retina (Hirschberg) ; multiple ruptures of the choroid (Meyhöfer, Franke, Pfalz) ; atrophy of the optic nerve (Blumenstock), etc., are found. Slight complications are rarely absent (de Wecker, Yvert). In Pfalz's case increasing myopia developed from stretching of the posterior pole of the eye. Eserin is without effect.

FIG. 783.—RUPTURES OF IRIS.

Parsons, Diseases of the Eye. Lacerations of the pupillary margin of the iris and dislocation of the lens following a blow. From a drawing by Holmes Spicer.

COOPER.—Ann. d'Oc., xxxiv, 1855. LAWSON.—Injuries of the Eye, London, 1867. *FRANKE.—A. f. O., xxxii, 2 ; xxxiii, 1, 1886. DE WECKER.—In G.-S., iv, 1876. VERMYNE. —T. Am. O. S., 1878. YVERT.—Traité, Paris, 1880. SCHIRMER.—K. M. f. A., xxviii, 1890. HIRSCHBERG.—Berliner klin. Woch., 1873. MEYHÖFER.—K. M. f. A., xv, 1877. PFALZ.— K. M. f. A., xxv, 1887. KAZAUROW.—C. f. A., xvi, 1892. POHLENZ.—Dissertation, Halle, 1891. LEVINSOHN.—A. f. A., xli, 1900. NEUBURGER.—Münchener med. Woch., 1901. DE SCHWEINITZ.—Ophth. Rec., 1901.

Radial ruptures of the iris (*rhexis iridis*) were mentioned by Demours, v. Ammon, Ried, Coccius, v. Arlt, and other early writers. All stages from simple ruptures of the sphincter are met with. When the globe is ruptured pieces may be torn out of the iris, so that a traumatic coloboma, sometimes as regular as an operative one, may be formed. Radial ruptures occur combined with iridodialysis, ruptures of the sphincter, traumatic mydriasis, etc. When the sclerotic is ruptured incarceration of the iris, retroflexion, etc., may be present.

HÖRING.—K. M. f. A., ix, 1871. HAYNES.—Brit. Med. Jl., 1872. GINZBURG.—Wjestnik oftalmologii, 1893.

Fissures of the iris.—Clefts in the iris, usually radial in direction, not extending into the pupil, are occasionally caused by blows with blunt objects (Schapringer, Paris, Lawson, Dufour, Clark). More rarely the fissures are not radial (Dohmen, Lawson), and sometimes they are multiple (Westphal). The condition is generally masked by hyphæma in the early stages, and may not be evident for 10—14 days (Clark, Westphal). The fissure is probably caused by pressure between the indented cornea and the resistent lens ; Praun likens it to rupture of the skin of a soft fruit when pressed between the thumb and a hard substance.

SCHAPRINGER.—A. f. A., xix, 1889. DOHMEN.—K. M. f. A., v, 1867. LAWSON.—Injuries of the Eye, London, 1867. DUFOUR.—K. M. f. A., viii, 1870. CLARK.—A. f. A., xxii, 1891. SCHMIDT.—In Nagel's Jahresbericht, 1889. WESTPHAL.—K. M. f. A., xxxiv, 1896.

Rupture of the pigment epithelium of the iris (*traumatic pigment coloboma*).—The retinal pigment epithelium of the iris may in rare cases alone be torn (Gelpke, Pohlenz, Boerma). This layer is doubtless less elastic than the stroma of the iris, and consequently ruptures more easily, though it must be remembered that some injuries may cause rupture of the stroma without the pigment layer (*cf.* Pfalz). In Gelpke's case there was a perforating wound and traumatic cataract.

GELPKE.—A. f. O., xxxiii, 3, 1887. POHLENZ.—Dissertation, Halle, 1891. BOERMA.—K. M. f. A., xxxi, 1893. PFALZ.—K. M. f. A., xxv, 1887.

Annular anterior synechia.—Purtscher records an extremely doubtful case of annular anterior synechia following a blow, attributing it to bruising of the pupillary margin by the cornea.

PURTSCHER.—C. f. A. xv, 1891.

Traumatic mydriasis and miosis (*traumatic iridoplegia*).—Immobility of the iris is often seen after severe blows upon the eye, rarely after lightning stroke (Leber). The pupil is generally moderately dilated, seldom contracted, still less often maximally dilated. In nearly all cases the pupil is irregular—pear-shaped, egg-shaped, transversely oval, or locally dilated in one part. Atropin acts very slowly, and causes only incomplete dilatation, perhaps due to absence of effective stimulation of the dilatator through the sympathetic fibres. In some

cases Schmidt-Rimpler has shown that the least dilated part before the use of atropin corresponds with the site of injury to the sclero-cornea. The pupil may be quite immobile or may react sluggishly to light. The mydriasis may pass off, usually very gradually, in the course of weeks or months, or may be permanent; it disappeared completely in two days in one case (Hirschberg).

Hyphæma and ruptures of the sphincter are very common accompaniments. The vision is usually much depressed, with photophobia; these symptoms generally pass off quickly and completely, especially the latter. There is often paresis or paralysis of accommodation, and the intra-ocular tension may be lowered (Nagel, Leplat). Rarely there is myopia, due to spasm of accommodation, or myopic astigmatism may be due to injury of the lens (Berlin). Opacity of the cornea and aqueous, vitreous hæmorrhage, subluxation of the lens, cataract, etc., may occur; more rarely retinal and choroidal hæmorrhages or rupture, detachment of the retina, macular changes, optic atrophy, etc.

Cases occur in which there are no visible ruptures of the sphincter, so that this, although an important factor, cannot be held to account for the condition *in toto*. Doubtless the paralysis is largely due to bruising and even rupture of the filaments of the oculo-motor and sympathetic nerves. Complete mydriasis would seem to demand paralysis of the oculo-motor with stimulation of the sympathetic fibres, which perhaps accounts for its rarity. There is some analogy in the physiological fact that stimulation of the sympathetic lasts much longer than that of other nerves (Praun).

Schmidt-Rimpler.—A. f. A., xii, 1883.   Berlin.—K. M. f. A., xi, 1873.   Hirschberg.—Klin. Beobachtungen, 1874.   Schlesinger.—In Nagel's Jahresbericht, 1874.   Leber.—A. f. O., xxviii, 3, 1882.   Marcus Gunn.—T. O. S., x, 1890.   Axenfeld, Dreyfus.—Deutsche med. Woch., 1906.

Ciliary Body.

Traumatic cyclitis—The typical signs of cyclitis, more especially the presence of precipitates on the back of the cornea ("k. p."), are seldom seen after contusions. Just as traumatic iritis in greater or less degree follows such injuries there can be no doubt that the ciliary body is also frequently involved. The haziness brought about by exudates in the aqueous must be attributed to traumatic iridocyclitis, and there is little doubt that the vessels of the ciliary processes often suffer severely. It must be remembered that precipitates on the cornea are evidence usually of a subacute or chronic process, and this is generally absent. The outcome of the injury to the ciliary body is the failure, more or less pronounced, of its normal functions, especially that of the secretion of the intra-ocular lymph, and this manifests itself unmistakably in a large number of cases by the diminution in the tension. Whether this is dependent upon paralysis of the nervous mechanism, rendered the more probable by the frequency of simultaneous paralysis of accommodation, but discounted by the negative evidence of experimental investigation (*v.* Vol. III, p. 982), or upon

direct injury to the secretory apparatus, must remain obscure in the present state of knowledge. The latter view has most in its favour.

Paralysis and spasm of accommodation.—It has already been pointed out that paresis or paralysis of accommodation (*traumatic cycloplegia*) or spasm of accommodation not infrequently accompany traumatic mydriasis or miosis respectively. The former is probably due to bruising and stretching of the motor nerve-fibres, as in the accommodative paresis of chronic glaucoma; it is likely that the fibres may be actually ruptured in some cases. Accommodative defects have been attributed also to variations in the blood supply in the more transitory cases, or to actual effusion of blood in the more severe. Myopia due to cramp of the ciliary muscle is of frequent occurrence (Berlin, Völckers, Schmidt-Rimpler, Just, Dehenne, Szili, Bettmann, v. Grolmann).

BERLIN, VÖLCKERS.—B. d. o. G., 1874. SCHMIDT-RIMPLER.—A. f. A., xii, 1883. JUST.—K. M. f. A., x, 1872. DEHENNE.—Ann. d'Oc., lxxxi, 1879. SZILI.—A. f. A., xiii, 1884. BETTMANN.—In Nagel's Jahresbericht, 1889. v. GROLMANN.—Monatschr. f. Unfall-heilk., 1896.

Rupture of the ciliary body.—Anatomical examinations of cases of rupture of the iris have shown that in a considerable proportion the

FIG. 784.—CONCUSSION INJURY.
Treacher Collins, Researches. Periphery of iris displaced backwards between lens and ciliary body.

FIG. 785.—CONCUSSION INJURY.
Treacher Collins, Researches. Iridodialysis and rupture of suspensory ligament and lens capsule on one side, rupture of fibres of ligamentum pectinatum, and splitting of ciliary muscle on the other side.

tear extended into the ciliary body (L. Müller, E. Schmidt). The condition cannot be diagnosed with accuracy clinically. The ciliary body may be separated in the whole circle from the sclerotic, remaining in

continuity with the choroid, or the ciliary body may be split so that the longitudinal fibres are left attached to the sclera at the anterior end, whilst the circular fibres are separated from them (Treacher Collins,

FIG. 786.—CONCUSSION INJURY.
Treacher Collins, Researches. Rupture of ligamentum pectinatum and splitting of ciliary muscle on both sides.

Buchanan). In one case it remained attached to the sclera only at the site of the rupture in the iris (L. Müller). A piece may be displaced,

FIG. 787.—CONCUSSION INJURY.
Treacher Collins, Researches. Rupture of ligamentum pectinatum and separation of iris and ciliary body from sclerotic, rupture of suspensory ligament and lens capsule on one side, with vitreous protruding forward between ciliary body and lens.

always torn away from the choroid in front of the ora serrata. Multiple ruptures occur, forming a definite traumatic coloboma.

FIG. 788.—CONCUSSION INJURY.
Treacher Collins, Researches. Dislocation of lens into anterior chamber, secondary glaucoma.

Transverse ruptures are relatively common in the posterior part. L. Müller considers that such ruptures may alone cause hyphæma.

L. Müller.—Die Rupter der Korneoskleralkapsel, Wien, 1895. E. Schmidt.—Disserta-
tion, Giessen, 1895. Treacher Collins.—T. O. S., xii, 1892 ; Researches, London, 1896.
Buchanan.—T. O. S., xxiii, 1903 ; xxvii, 1907.

CHOROID.

Hæmorrhage and detachment.—Rupture of choroidal vessels may
cause small intra-choroidal hæmorrhages, visible ophthalmoscopically, or
subchoroidal or retinal hæmorrhages of greater or less degree ; the
retina may be perforated, bleeding into the vitreous resulting. Blood
may pass through a tear in the retina near the ora serrata and pass
through the zonule into the anterior chamber (Zander and Geissler).

Siegrist describes four cases of contusion of the eye with yellow
coloration around the disc and in the macular region, sharply defined
in contour. Pigmentation of the retina in the affected areas follows.
On the ground of Wagenmann's experiments (v. Vol. II, pp. 593,
606) Siegrist attributes the condition to rupture of posterior ciliary
arteries.

Detachment of the choroid from subchoroidal hæmorrhage is
commonly seen in the pathological laboratory, but the clinical picture
described and figured by v. Graefe and Liebrich is seldom seen.
Clinical observations are reported of three cases after contusion
(v. Michel, Walter, Sous), others after cataract extraction (Knapp,
Groenouw, de Wecker, Berger, Hay, Elschnig, Marshall). Anatomical
observations, especially after extraction and in phthisis bulbi, were made
long ago (v. Ammon, Hulke, Bowman, Cooper, and others). Detach-
ment after cataract extraction, iridectomy, etc., may be due to escape
of aqueous into the subchoroidal space (Fuchs, Demaria, Fischer).

The ophthalmoscopic picture of detachment of the choroid, so
seldom seen, is open to doubt. There is said to be apparent detach-
ment of the retina, which does not move on movement of the eye, is
reddish brown or yellow, often covered with hæmorrhages. It was
shallow in the cases of v. Michel and Sous, prominent in that of
Walter. In the two former the choroid became replaced and vision
was restored. The diagnosis from sarcoma of the choroid depends
largely upon the history. v. Michel attributes the bleeding to rupture
of a large vessel at the posterior pole ; the retinal hæmorrhages are
ascribed to the stretching of the retina and impediment to the return
of venous blood.

Siegrist.—Mittheil. aus d. Kliniken u. med. Instituten der Schweiz, Basel, 1895.
Wagenmann.—A. f. O., xxxvi, 4, 1890. Knapp.—Die intra-ocularen Geschwülste, 1868.
Groenouw.—A. f. A. xx, 1889. de Wecker.—In G.-S., iv, 1876. Berger.—Bayer. Intelli-
genzbl., 1878. Hay.—In Nagel's Jahresbericht, 1883. Elschnig.—A. f. A., xxiii, 1892.
v. Michel.—K. M. f. A., xvi, 1878 ; Lehrbuch. Walter.—Dissertation, Würzburg, 1883.
Sous.—In Nagel's Jahresbericht, 1892. Story.—Brit. Med. Jl., 1881. Mules.—T. O. S.,
xiii, 1893. Marshall.—T. O. S., xvi, 1896. Fuchs.—A. f. O., li, 2, 1900. Demaria.—
K. M. f. A., xlii, 1904. Fischer.—A. f. A., lvii, 1907. Terson.—A. d'O., xxvii, 1907.

Rupture of the choroid is not uncommon as the result of indirect
injury. It was first described clinically by v. Graefe (1858), and
subsequent cases were collected by Caillet (1869), Knapp (1870), de
Wecker (1877), Achard (1877). v. Ammon had already described a

case anatomically. Blows directly on the front of the eye are most disposed to cause choroidal rupture. Unusual causal injuries are reported by v. Ammon, injury to the mouth with fracture of the inner wall of the orbit ; Adamuk, gunshot ; Mannhardt, severe concussion of the whole body ; Benson, fall from a horse ; Schmidt-Rimpler, forceps delivery in a baby.

Hughes, who collected the recorded cases, found temporal rupture in 82 per cent., nasal in 14 per cent., horizontal in 4 per cent. The rupture is almost always near the posterior pole, concentric with the disc, and between it and the macula. As soon as ophthalmoscopic observation is possible it appears as a yellow curved streak, bordered or covered in places by blood with more or less pigment. After the blood has become absorbed the sickle-shaped defect is faint yellow or white, broadest in the middle ($\frac{1}{2}$—$\frac{1}{3}$ p.d.), 2—3 p.d. in length, pointed at the ends, sometimes edged with pigment. Probably the lamina fusca is torn, accounting for the white, not bluish, appearance (de Wecker). Usually only the pigment epithelium of the retina suffers, but rarely the retina is also ruptured (Cowell, Hirschberg, Genth, Bäuerlein, Magnus, Lawson, Hughes). The slight hæmorrhage is due to the intra-ocular pressure supporting the vessels. The surrounding retina is at first œdematous.

Atypical ruptures may be enumerated : extreme length (Mannhardt), forked extremities (Aub), horizontal (Mauthner, Teillais, Hirschler)— triangular (Hughes), choroid and retina (Hutchinson), vertical (Mannhardt, de Wecker), nearly surrounding the papilla (Knapp, Ginzburg), through the macula (Adamük, Vossius), very peripheral (Saemisch—probably direct rupture), etc. There are often multiple ruptures, when the central are usually the larger, often with radial tears between ; the more peripheral are yellower, probably involving only the deeper layers of the choroid. Cases are recorded by Polano, Genth, Talko, Hersing, Vossius, Banister, below the papilla, Benson, Fage, Teillais, Aub, central and peripheral (Hoor).

There are often complications—traumatic mydriasis, cycloplegia, contracted field (Pohlens—due to rupture of nerve-fibres), dislocation of the lens, retinal hæmorrhages (Vossius, Knapp, Mannhardt), detachment of the retina, etc.

Cicatrisation may be followed by atrophy and detachment of the retina, changes at the macula, etc. Streatfeild records a case of secondary glaucoma with cupping of the disc.

Direct rupture of the choroid is rare and always situated at the periphery. The tears are usually large and irregular, and much broader than those in indirect rupture, exposing the sclerotic (de Wecker). The retina may be torn or detached, and is generally studded with hæmorrhages. This injury often follows gunshot wounds (Schröter, Buard, Bruha) ; it has been observed only once as a birth injury (Schmidt-Rimpler).

The mechanism of indirect rupture of the choroid has been much discussed. Kern and v. Seidlitz attribute it to *contrecoup*, a view which is opposed to the laws of hydrodynamics, since the force should be equally distributed in all directions throughout the fluid mass. Knapp

and Aub point out the analogy with fractures of the skull by *contrecoup*, but the analogy is very fallacious (v. Arlt, Geissler). Becker attributes the injury to invagination of the optic nerve head by the blow— a highly improbable theory when the mobility of the nerve and the opposition of the intra-ocular pressure are taken into account. Saemisch considers that the fixing of the choroid to the sclerotic by the posterior ciliary vessels may be an important factor; this view is supported by de Wecker, but Knapp points out that in all conditions of stretching the choroid and sclerotic are equally affected and retain their normal relative positions. v. Arlt, bringing choroidal rupture into line with scleral rupture, advanced the view that flattening of the globe by the blow causes stretching of the choroid, which gives way at right angles to the direction of extension, and at the site where it is least firmly attached to the sclera. Berlin, followed by Geissler and Hirschberg, thought that the rupture was caused by forcing the globe against Tenon's capsule and the orbital contents, supported as these are by the bony walls. Hülse attributed the size and shape of the tear to contraction of the ciliary muscle—a theory of merely historical interest. Franke conjectures that the recoil after the blow acts least on the parts surrounding the nerve head and thus causes the rupture, but there is no reason to suppose that the force of the recoil is unequally distributed (Hughes, v. Michel). Hughes and v. Michel introduce a new element into the discussion, viz. rotation of the globe. If the blow is directed more or less tangentially the eye will rotate whilst the intra-ocular pressure is simultaneously raised. The rotation is stopped by a sudden jerk when the optic nerve is put on the stretch. The escape of the retina is attributed to its greater elasticity. This theory is accepted by Parisotti.

Experimental observations have been made by Berlin and v. Seidlitz on rabbits, Hillenkamp on dogs, Caillet on hares, and others.

v. Graefe.—A. f. O., i, 1, 1854. Caillet.—Thèse de Strasbourg, 1869. Knapp.— A. f. A., i, 1869. de Wecker.—Traité. Achard.—Thèse de Paris, 1877. Mauthner.— Lerhbuch der Ophthalmoscopie, Wien, 1868. v. Ammon.—A. f. O., i, 2, 1855. Ai amük.— C. f. A., ii, 1878. Mannhardt.—K. M. f. A., xiii, 1875. Benson.—Brit. Med. Jl., 1883. Schmidt-Rimpler.—Die Erkrankungen des Auges, etc., Wien, 1905. de Wecker.— In G.-S., iv, 1876. Aub.—A. f. A., ii, 1870. Teillais.—Ann. d'Oc., lxxvii, 1877. Hirschler—In Nagel's Jahresbericht, 1887. *Hughes.—A. f. O., xxxiii, 3, 1887. Hutchinson.—R. L. O. H. Rep., xii, 2, 1888. Vossius.—K. M. f. A., xxi, 1883. Saemisch.— K. M. f. A., v, 1867. Hoor.—Wien. med. Woch., 1886. Polano.—Dissertation, Kiel, 1897. Genth, Schröter, Talko.—K. M. f. A., ix, 1871. Hersing.—K. M. f. A., x, 1872. Banister.—C. f. A., xviii, 1894 (Suppl). Fage.—In Nagel's Jahresbericht, 1894. Pohlenz. —Dissertation, Halle, 1891. Cowell.—R. L. O. H. Rep., vi, 4, 1869. Hirschberg.—Berlin. klin. Woch., 1870, 1875. Bauerlein.—Blätter f. Heilw., ii, 1871. Magnus.—K. M. f. A., xxv, 1887. Lawson.—Injuries of the Eye, London, 1867. Streatfeild.—In Knapp. Buard.—Thèse de Montpellier, 1885. Bruha.—Dissertation, Kiel, 1889. Kern.— Deutsche militärärztl. Zeitschrift, xv. v. Seidlitz.—Dissertation, Kiel, 1873. Geissler.— Schmidt's Jahrbuch, 1873, 1874. Becker.—K. M. f. A., xvi, 1878. Saemisch.— K. M. f. A., xxiv, 1886. v. Arlt.—K. M. f. A., xii, 1874. Berlin.—K. M. f. A., xi, 1873. Hülse.—Dissertation, Kiel, 1878. Franke.—A. f. O., xxx, 2, 1884. v. Michel.— Lehrbuch, Wiesbaden, 1890. Parisotti.—Rev. gén. d'O., 1887. Haass.—Z. f. A., xi, 1904. G. W. Thompson.—T. O. S., xxiv, 1904. Beck.—A. f. A., lvi, 1906. Kröner.— A. f. A., lv, lvi, 1906.

LENS.

Contusion cataract (*indirect traumatic cataract*).—Contusion of the eye may lead to cataract without direct rupture of the lens capsule. Two groups of cases may be distinguished : (1) those in which there is a demonstrable rupture of the capsule ; (2) those in which no such rupture can be demonstrated. In the first group total cataract usually ensues ; in rare cases the wound in the capsule heals, and the opacity remains localised, sometimes even diminishing in extent. The second group are to be attributed for the most part to circulatory disturbance in the ciliary body (O. Schirmer) ; only rarely is it possible that invagination of the wall of the globe leads to a sort of massage cataract.

The rationale of rupture of the capsule may be described as follows : The blow upon the eye leads to diminution of the antero-posterior diameter with increase of the equatorial diameter. The zonule is stretched, resulting most commonly in rupture and dislocation of the lens. The zonule may, however, escape rupture, the posterior capsule of the lens giving way owing to its tenuity (R. Schirmer). Both zonule and posterior capsule may of course be ruptured. The mechanism of rupture of the anterior capsule is probably different. Here the object indents the cornea, which is forced up against the iris and lens. Hence it is not uncommon to find a line of pigment near the slit in the capsule (Schirmer). This explanation is the more probable because the rupture shows no tendency to uniform direction, the direction being determined by the incidence of the blow. The first theory explains such cases as those of Knapp and Aub, the second those of Quadri, Becker, Bresgen, Landesberg, Liebrecht, Lenz, and others. Dislocation of the lens associated with rupture of the anterior capsule is chiefly due to the displacement backwards of the aqueous. Change in the shape of the lens is most readily induced in young people, thus accounting for the prevalence of capsule rupture in them. Traumatic cataract from pressure of forceps during birth is recorded by Peck.

In the absence of rupture of the capsule the opacity of the lens may be transient, *e. g.* 36—40 hours (Magnus), hours or days (Schirmer's experiments). There may be a star-shaped opacity in the posterior cortex (Fuchs), not improbably due to transient disturbance of nutrition through the ciliary body. In Fuchs's case there was also iridiodialysis, and the cataract cleared up almost completely in eight weeks. Care must be taken to distinguish true intra-lenticular opacity from deposits of blood or exudate upon the anterior capsule in con-tusion cases ; the prognosis is more favourable in the latter. There is no doubt that contusion may lead to total and persistent opacity of the lens (Praun). Occasionally there are deposits of iris pigment on the anterior capsule (Beer, v. Arlt).

The capsule probably ruptures most frequently near the equator of the lens (Becker, Treacher Collins, associated in this case with rupture of the suspensory ligament). Rupture at the posterior pole, causing posterior lenticonus, is rare (Treacher Collins, Chiari). The rupture

may be in any direction, or star shaped. The subsequent course is that of a traumatic cataract caused by perforation, the lens substance

FIG. 789.—TRAUMATIC CATARACT.

Treacher Collins, T. O. S., xi.   Rupture of posterior capsule of lens and extrusion of lens substance through aperture, from a blow with a stone.

being gradually more or less completely absorbed (Werner, Becker, Bresgen, Letenheuer, etc.).   The lens nucleus may become dislocated into the anterior chamber (Quadri, Testelin, Ulrich), as occurs

sometimes after discission. The rupture of the capsule may close, being followed generally by complete opacification of the lens (Bresgen, Hosch), or rarely the opacity remains localised, sometimes clearing up to some degree (Bresgen, Landesberg, Liebrecht). The latter result is much more likely to occur in concussion cataract than in cases of traumatic perforation of the capsule.

The most frequent complication is partial or complete dislocation of the lens, though injuries to other parts of the eye are not uncommon.

LENZ.—C. f. A., xxi, 1897. WERNECK.—v. Ammon's Zeitschrift, iv. O. SCHIRMER.—Dissertation, Greifswald, 1887. SCHLÖSSER.—Exp. Studie über die traumat. Katarakt, München, 1887. FUCHS.—Wien. klin. Woch., 1888. R. SCHIRMER.—K. M. f. A., xxviii, 1890. MAGNUS.—Deutsche med. Woch., 1888. TREACHER COLLINS.—T. O. S., xi, 1891. LAWFORD.—Ophth. Rev., vi, 1887. HOSCH.—A. f. A., xx, 1889. QUADRI.—In Zander and Giessler, p. 358. KNAPP.—A. f. A., i, 1869. AUB.—A. f. A., ii, 1870. PECK.—Med. News, 1898. COOPER.—On Injuries of the Eye, London, 1859. BECKER.—In G.-S., v, 1877. BRESGEN.—A. f. A., x, 1881. ULRICH.—K. M. f. A., xx, 1882. SCHÖLER.—Jahresbericht, 1878. LANDESBERG.—K. M. f. A., xxiv, 1886. LIEBRECHT.—B. z. A., xviii, 1895. HUTCHINSON.—Ophth. Rev., viii, 1889. MERZ-WEIGANDT.—C. f. A., xxiv, 1900. BÖSE.—Z. f. A., ix, 1903. ZUR NEDDEN.—Z. f. A., xi, 1904. CHIARI.—Ann. di Ott., xxxiii, 1904. PARDO.—A. di Ott., xiii, 1905. WAGENMANN, v. HEUSS.—B. d. o. G., 1905.

Stretching of the suspensory ligament.—Cases of myopia following contusion of the eye have been attributed to stretching of the zonule (Aub, v. Arlt, Schiess, Knapp, Krienes), though on quite insufficient grounds. It is more probable that the refractive error is due to spasm of the ciliary muscle.

AUB.—A. f. A., ii, 1870. v. ARLT.—Die Verletzungen des Auges, Wien, 1875. SCHIESS.—K. M. f. A., xix, 1881. KNAPP.—A. f. A., xii, 1883. KRIENES.—Festschrift des Fr. Wilhelm Instituts, Berlin, 1895.

Dislocation of the lens.—Partial or complete rupture of the suspensory ligament causes dislocation of the lens, also partial or complete. It is possible that there may be slight partial rupture of the zonule without any demonstrable displacement of the lens. Some cases of lenticular astigmatism following a blow have been explained upon these grounds.

Rupture of the zonule of greater extent causes *subluxation of the lens*. The displacement of the lens is twofold : towards the uninjured side (dislocatio ad latus), and on its equatorial axis (dislocatio ad axem), the latter being due to the curvature of the surfaces and the curvature of the patellar fossa. The anterior chamber is shallower, owing to displacement forwards of the iris, on the side towards which the lens is displaced. On the opposite side the anterior chamber is abnormally deep and the lens is abnormally movable, as shown by tremulousness which is transmitted to the iris (iridodonesis). Myopia, astigmatism, and greater or less cycloplegia accompany the release of the lens capsule from the anchoring fibres of the suspensory ligament. The edge of the lens may cross the pupil, causing uniocular diplopia. The lens may remain clear or become cloudy.

Subluxation may pass into complete dislocation, especially in elderly people in whom the suspensory ligament is likely to be degenerated. The pressure of the subluxated lens upon the iris and

ciliary body may lead to iridocyclitis. More to be feared is secondary glaucoma, induced by obliteration of the angle of the anterior chamber. Owing to the similarity of curvature of the equator of the lens and of the ciliary border of the iris a considerable part of the angle is
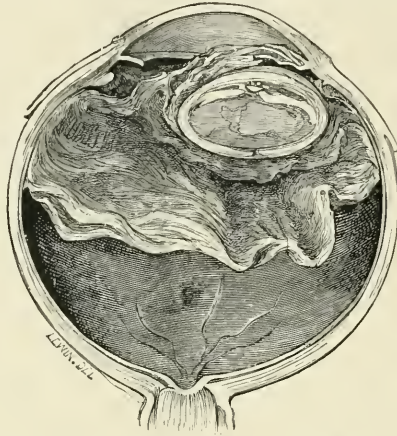
FIG. 790.—DISLOCATION OF THE LENS. × 2½.
R. L. O. H. Museum, Series I, Sub-series A, No. 29. Lateral displacement of lens, showing the vitreous passing round its margin into the anterior chamber.

occluded, and this may be further diminished by the inflammatory reaction.

SCHÖLER.—Jahresbericht, 1875. PFLÜGER.—K. M. f. A., xiii, 1875.

Total rupture of the suspensory ligament causes complete dislocation or *luxation of the lens*. It is not essential in these cases that the zonule should be absolutely severed in the whole of its circumference, though this also occurs. Förster's theory of the sudden displacement backwards of the aqueous best explains the rupture of the suspensory ligament. Blows upon the limbus cause luxation towards the site of incidence. In such laterally directed blows the mechanism suggested by Förster does not fully elucidate the displacement. It is probable that in these, and, indeed, in most other cases, there is some inherent weakness of the zonule, and this view is supported by the preponderance of elderly patients amongst the recorded cases. Many other cases exhibit high myopia, staphylomata, fluidity of the vitreous, old-standing cyclitis, congenital ectopia lentis, etc., conditions which also point to predisposition owing to degenerative changes or congenital defects.

The lens may be dislocated forwards into the anterior chamber, either partially or completely, backwards into the vitreous, or may be freely movable, changing its position from time to time.

Dislocation of the lens into the anterior chamber occurs most commonly in disorganised eyes in which the lens is more or less shrunken and the zonule stretched and degenerated. It may, however, occur with a clear lens of normal size. The diagnosis is not always easy in these

cases, and the condition may nearly resemble that of blood staining of the cornea (q. v.)

The anterior chamber is deep, the iris being displaced backwards. Owing to the release from the zonule the lens is more globular than when *in situ*; if the lens is clear and any vision persists there is high myopia (Pflüger), due to the displacement forwards and to the increased curvature of the lens surfaces. If the lens is small and shrunken there may be little reaction, but even in these cases secondary glaucoma may ensue at any moment, or the eye may be lost by plastic iridocyclitis. It is said that iridocyclitis is more likely to occur in those cases in which the zonule is not completely severed, owing to dragging on the ciliary processes (v. Graefe). The zonule may be intact (Becker, Hess). The danger of secondary glaucoma is increased if the lens is large, since its presence in the anterior chamber greatly impedes the outflow of lymph at the filtration angle, and inflammatory reaction in the iris may cause further blockage. Cases are reported in which the instillation of eserin has set up or aggravated glaucoma by causing the iris to contract firmly against the back of the lens (Priestley Smith). Beccaria reports a case in which the patient could produce glaucoma at will by bending his head in certain directions, especially forwards. Sympathetic ophthalmia has been reported as a sequel (Dermett).

The lens may be only partially displaced into the anterior chamber. It is then firmly grasped by the sphincter iridis, and intense congestion of the iris is set up. Usually the cornea becomes cloudy rapidly, and there is difficulty in diagnosing the condition. Riedel records a case in which the posterior surface of the lens faced forwards, and the nasal portion was prolapsed into the anterior chamber.

Dislocation of the lens into the vitreous is the commonest displacement. The anterior chamber is very deep, the iris tremulous. The lens usually falls to the lowest part of the vitreous chamber, and may be visible ophthalmoscopically or by oblique illumination. It may move on movement of the eye. Only rarely is it absorbed (*e.g.* Snell, Augstein and Ginsberg), since the capsule is often intact. It may remain clear, when it is seen only with great difficulty; in these cases it derives nutrition from the lymph in the vitreous. Jaeger saw the lens clear in the vitreous after thirty years; in Guépin's case it became opaque after twenty-five years. Dislocation of the clear lens into the vitreous usually occurs in highly myopic eyes. The contents only of the capsule may be dislocated (Leber, Wagenmann, Habben), chiefly after purulent changes around the lens.

There is great danger of plastic cyclitis being set up, as is well known from the results of reclination. The tension usually rises, then falls, and phthisis bulbi follows. The eye may, however, remain quiet (Aub, Andrew, and others).

Hock reports a case of oblique dislocation of the nucleus only, much open to doubt.

Free mobility of the lens occurs in some cases of total luxation (Heymann, Noyes, Nettleship, Dub, and others). It usually occurs in congenitally small or in shrunken lenses.

Complications are often present in luxation of the lens, such as traumatic mydriasis, iridodialysis, rupture of the choroid, hæmorrhage in various situations, etc. Schmeichler reports rupture of the ciliary body, Berger detachment of the retina, etc.

HIMLY.—Ophth. Beobachtungen, 1801. v. AMMON.—Z. f. O., i, 1831. v. GRAEFE.— A. f. O., i, 1, 1854; ii, 1, 1855. E. MÜLLER.—A. f. O., viii, 1, 1861. TALKO.—A. f. A., ix, 1880. THEOBALD.—T. Am. O. S., 1881. BENSON.—Brit. Med. Jl., 1882. FÖRSTER.— B. d. o. G., 1887. WILLIAMS.—T. Am. O. S., 1875. BADAL.—Nagel's Jahresbericht, 1878. PFLÜGER.—K. M. f. A., xiii, 1875. PRIESTLEY SMITH.—Ophth. Rev., v, 1886; Glaucoma, London, 1891. BECCARIA.—Ann. di Ott., xxii, 1893. LAWFORD.—R. L. O. H. Rep., xi, 1887. RAMPOLDI.—Nagel's Jahresbericht, 1882. RIEDEL.—Dissertation, Greifswald, 1894. SNELL.—Ophth. Rev., i, 1882. GUÉPIN.—Ann. d'Oc., xvi, 1846. JAEGER.—Schmidt's Jahrbuch, v. AUB.—A. f. O., ii, 1, 1871. ANDREW.—Brit. Med. Jl., 1882. HOCK.—Realencyclopädie der ges. Heilk., i. HEYMANN.—Ann. d'Oc., xlviii, 1861. NOYES.—A. of O., i, 1, 1869; B. d. o. G., 1884. NETTLESHIP.—T. O. S., i, 1881. DUB.—Wiener med. Woch., 1888. SCHMEICHLER.—Wiener med. Woch., 1887. BERGER.—A. f. A., xv, 1885. PENET. —Dissertation, Lyon, 1884. HENKE.—Dissertation, Strassburg, 1893. WALKER.—Lancet, 1892. ZIMMERMANN.—A. of O., xxii, 1893. CLARK.—T. Am. O. S., 1894. E. v. HIPPEL, EVERSBUSCH.—Münchener med. Woch., 1895. AUGSTEIN AND GINSBERG.—C. f. A., xx, 1896. DORSCH.—Dissertation, Marburg, 1900. STOEWER.—Z. f. A., v, 1901. *HESS.—In G.-S., vi, 2, 1905 (Bibliography).

Subconjunctival dislocation of the lens has already been referred to

FIG. 791.—SUBCONJUNCTIVAL DISLOCATION OF LENS.
Henderson, T. O. S., xxiv. Showing scleral rupture, prolapse of iris, and subconjunctival dislocation of the lens, which is undergoing absorption.

incidentally (*v.* p. 1138). The lens passes through the root of the iris (L. Müller), usually carrying the iris with it, and not through the pupil, as suggested by Massie. The lens is generally dislocated in its capsule, though the latter is often ruptured. v. Graefe records a curious case in which only part of the cortex passed under the conjunctiva, the remainder, with the nucleus, staying in the capsule within the eye. L. Müller reports a similar case. Mitvalski found the capsule remain-

ing in the eye only once in thirteen cases. In one of his cases the capsule formed a cyst communicating with the anterior chamber. In Hulke's case the cornea was split, so that the lens lay between its layers, extending partly over the limbus. The lens may be at some distance from the site of scleral rupture (Briolat). Usually the lens fibres are rapidly absorbed, but they may long remain almost unaltered, *e.g.*, fifteen years (Vieusse), eighteen years (Treacher Collins), probably owing to an intact capsule. Calcareous deposits may mark its site (v. Arlt), or a cyst, which may discharge periodically (Ansiaux).

Incarceration of the lens in the wound is recorded by Jaeger, Lederle, Sichel, Fano, Mercanti, Treacher Collins.

Dislocation in Tenon's capsule has been reported by Bowman, Weeks, Chisholm, Nuel, Wadsworth, Montagnon (between external and superior recti), and Schlodtmann (between external rectus and superior oblique).

Sympathetic ophthalmia following subconjunctival dislocation of the lens is reported by Treacher Collins.

SACHS.—A. f. A., xx, 1899. L. MÜLLER.—Ueber Ruptur der Korneoskleral kapsel durch stumpfe Verletzung, Leipzig. 1895. MASSIE.—Thèse de Paris, 1875. v. GRAEFE.— A. f. O., i, 1, 1854; iii, 2, 1857. ALT.—A. f. A., vi, 1877. MITVALSKI.—A. d'O., xvii, 1897. BRIOLAT.—Thèse de Paris, 1879. RIEGEL.—Dissertation, München, 1888. VIEUSSE.—Rec. d'O., 1879. TREACHER COLLINS.—R. L. O. H. Rep., xii, 1889. JAEGER.—Staar u. Staaroperationen, 1854. LEDERLE.—K. M. f. A., xiii, 1875. SICHEL.—Iconographie, pl. xix. FANO.—Ann. di Ott., ix, 1880; Jl. d'Oc., viii, 1880. FALCHI.—Ann. di Ott., xiv, 1883. MERCANTI.—Ann. di Ott., xx, 1891. CHISHOLM.—Amer. Jl. of O., ix, 1892. WADSWORTH.— Amer. Jl. of O., ii, 1885. MONTAGNON.—A. d'O., vii, 1887. SCHLODTMANN.—A. f. O., xliv, 1, 1897. ANDRÉ.—Ann. d'Oc., lxviii, 1872; lxxii, 1874. ZEHENDER.—K. M. f. A., xiii, 1875. WORDSWORTH.—R. L. O. H. Rep., x, 1881. PAON.—Ann. d'Oc., cxxxii, 1904. HENDERSON. T. O. S., xxiv, 1904. PURTSCHER.—Hirschberg's Festschrift, Leipzig, 1905. HESS.—In G.-S., vi, 2, 1905. CANTONNET.—A. d'O., xxvi, 1906.

VITREOUS.

Intra=vitreous hæmorrhage.—Hæmorrhage into the vitreous is of very common occurrence as the result of contusions of the eye. The blood is derived from ciliary, retinal, or choroidal vessels. If these are already diseased the severity of the contusion may be very slight : indeed, apparently spontaneous bleeding not infrequently occurs. The blood is not absorbed so rapidly as from the anterior chamber, taking 6—8 weeks or more. Permanent vitreous opacities are likely to remain, the vitreous itself becoming degenerated and fluid. In relatively rare cases the blood organises, so-called " retinitis pro-liferans " occurring (*v.* Vol. II, p. 609).

RETINA.

Traumatic œdema of the retina (*commotio retinæ*).—The term " commotio retinæ " was applied to all cases of depression of vision following severe injuries to the head or eye, on the analogy of commotio cerebri. Greater accuracy of the methods of investigation has reduced the scope of the term to cases of œdema of the retina following blows. Berlin (1873) first attacked the problem in an

exhaustive manner from both the clinical and experimental standpoints. He carefully eliminated from this category the impairment or loss of vision which follows fracture of the base of the skull, for these cases are generally due to retrobulbar injury or section of the optic nerve. v. Hoelder showed that most fractures of the skull pass through the base, and that of these more than half involve the optic foramen and cause rupture of the nerve.

Commotio retinæ (Berlin) was first described by Hirschberg (1875), then by Leber (1877), Nettleship (1880), Knapp (1881), Schmidt-Rimpler (1883), Haab (1885), Dimmer (1885). There are later papers by Siegfried, Linde, and others.

Œdema of the retina is not uncommon after a contusion of the eye, and the mechanism of its production is similar to that of rupture of the choroid, which may be also present. At the region corresponding with the incidence of the transmitted force there is a greyish or whitish area in the retina, and a similar though smaller area is found at the macula, often separated from the larger area by normal retina (Haab). After blows directed straight backwards the circumpapillary region is chiefly affected. The size of the area varies, and white streaks may radiate from its edges. The vessels appear darker than normal and show some convolution. The macula looks abnormally red by contrast (vide infra, " Hole " in the retina). The sensibility of the retina is greatly depressed in the early stages (Schmidt-Rimpler), the central vision being diminished out of proportion to the peripheral (Berlin), though contraction of the field must be regarded as a regular concomitant (Ostwald, Makrocki, Dimmer). The cloudiness of the retina develops during the first 24—48 hours, and may disappear in from 3—4 days. Permanent loss of function is to be attributed to complicating injury of the macula or retina, which is of frequent occurrence (vide infra).

Miosis, mydriasis, myopia, myopic lenticular astigmatism, hæmorrhages, etc., are common complications, as well as hæmorrhage into the nerve sheath, and even fracture of the optic foramen. Ancke reports unilateral discoloration of the disc with retained normal function.

Haab gives the following statistics : In 167 cases of contusion of the eye in which ophthalmoscopic examination was possible 82·6 per cent. showed no changes, 12·5 per cent. œdema of retina and macula, 4·7 per cent. œdema of macula only. Of the 12·5 per cent. sixteen cases cleared up entirely, with normal or nearly normal vision. In five cases pigmentation of the macula followed, visible 1, 3, 4, 8, and 19 days respectively after the injury. The prognosis in these cases is much worse, the best vision being $\frac{6}{60}$, and in one case optic atrophy, in another choroidal changes, ensued. Lawford describes extensive retinal pigmentation and choroidal atrophy.

Berlin attributed the œdema to extravasation of blood between the choroid and sclerotic, such as he observed in rabbits. This view is untenable on the grounds that blood is never extravasated between the retina and choroid in these cases, as might be expected at least as an occasional event, and retinal œdema does not accompany choroidal

rupture ; moreover, the rapid recovery is against the presence of a hæmorrhage. Hirschberg considered it probable that the blow caused local reflex vaso-constriction, leading to transitory blindness, followed by increased permeability of the vessels, resulting in œdema. Berlin's experiments support the view of a temporary anæmia. Makrocki invokes molecular changes in the nerve-fibres as a result of the anæmia. Ostwalt and Haab state that the retinal vessels participate in the anæmia. Vaso-constriction of pathological origin in other parts of the body is followed by vaso-dilatation, which increases the tendency to transudation, and this is likely to be very marked in the macular region, where the chorio-capillary network is specially fine. Denig's experiments on animals support on the whole Berlin's findings, and his conclusions have in turn been confirmed by Bäck. Denig considered that the vitreous was forced against the retina, leading to separation of the expanded ends of Müller's fibres, rupture of the internal limiting membrane, fluid being squeezed into the nerve-fibre layer. Paralysis of the retinal and choroidal vessels caused transudation, the fluid in the latter instance being situated between the rods and cones. In the more severe cases active dislocation of the structures in the layers of the retina is probable. Tepljaschin found the maximum accumulation of fluid in the inner nuclear layer. That the essential cause of the œdema lies in the condition of the vessels is shown by Wagenmann's experiments (v. Vol. II, pp. 593, 606) and by the macular œdema which occurs in cases of division of the optic nerve anterior to the site of entry of the central vessels (v. infra, p. 1184). Further, there are other cases of commotio retinæ without direct injury to the globe, which must be explained on similar grounds (e. g. Kammann).

BERLIN.—K. M. f. A., xi, 1873; in G.-S., vi, 1880; Berliner klin. Woch., 1881. v. HOELDER.—B. d. o. G., 1879. NETTLESHIP.—Lancet, 1880. KNAPP.—A. f. A., x, 1881. HERDEGEN.—A. f. A., x, 1881. SCHMIDT-RIMPLER.—A. f. A., xii, 1883 ; K. M. f. A. xxii, 1884. HIRSCHBERG, OSTWALT.—C. f. A., x, 1887. HAAB, SIEGFRIED.—B. z. A., xxii, 1898. MAKROCKI.—A. f. A., xxiv, 1892. DENIG.—A. f. A., xxxiv, 1897; A. f. O., xlvii, 3, 1899. BÄCK.—A. f. O., xlvii, 1, 1898. TEPLJASCHIN.—Nagel's Jahresbericht, 1893. WAGENMANN. —A. f. O., xxxvi, 4, 1890. KAMMANN.—Dissertation, Kiel, 1893. HAAB.—B. d. o. G., 1888 ; Atlas der Ophthalmoscopie, pls. 49, 50. DIMMER, ANCKE.—C. f. A., ix, 1885. LINDE.— C. f. A., xxi, 1897. HUTCHINSON, JR.—T. O. S., ix, 1884. LAWFORD.—T. O. S., xxii, 1902. BICKERTON,—T. O. S., xxiv, 1904. FRIDENBERG.—A. f. A., lii, 1905. LOHMANN.—A. f. O., lxii, 1906; K. M. f. A., xliv, 1906.

Retinal hæmorrhage.—Small hæmorrhages from the retinal vessels are common after contusion of the eye. They are generally single, but multiple hæmorrhages often occur. They vary in appearance according to their situation in the retina, those in the nerve-fibre layer being flame shaped, those in other parts round or angular. Traumatic hæmorrhages are usually situated in the deeper layers. A large subhyaloid or pre-retinal hæmorrhage may occur in the macular region, rapidly becoming more or less hemispherical in shape owing to the action of gravity. It becomes absorbed without much or any impairment of vision. Rarely two or more such hæmorrhages may be present in the same eye (Holmes Spicer and others). They have been investigated anatomically by Fisher, v. Benedek, and others.

Intra-retinal hæmorrhages take one to two months for complete

absorption, and often leave white or yellow, less frequently pigmented, spots of degeneration to mark their sites. Very extensive hæmorrhages may cause detachment of the retina, and may be followed by pigmentation and atrophy of the optic nerve. Hæmorrhage at the macula is naturally of worst prognosis as to sight. The black pigment spots may be due to proliferation of retinal epithelium or to hæmatogenous pigmentation (Hirschberg, Hersing, and others).

Hæmorrhages in and under the retina are common in shot wounds of the orbit, though the globe may escape direct injury. I have reported one such case and many others are on record.

HOLMES-SPICER.—R. L. O. H. Rep., xiii, 3, 1892. FISHER.—R. L. O. H. Rep., xiv, 2, 1896. V. BENEDEK.—A. f. O., xlii, 1906. PARSONS.—T. O. S., xxvi, 1906.

Retinal hæmorrhages are not infrequently found immediately after normal (Königstein, Schleich, Naumoff) and artificially assisted parturition (Truc, Nettleship, Thomson and Buchanan, Wolff). Königstein found them in 10 per cent. of births (29 in 281 cases), and even in children born at seven and eight months; Schleich in 32 per cent. (49 in 159 cases) ; E. v. Hippel in 42 per cent. examined anatomically, and Naumoff in 26·5 per cent., but none in premature births. Naumoff, who investigated the eyes of forty-seven new-born children microscopically, found them chiefly in the periphery in the nerve-fibre and ganglion cell layers, and also macular in the inner nuclear layer. He also saw three cases of choroidal hæmorrhage, one at the macula with circumscribed detachment of the retina. Königstein considers that they arise during the first act of respiration, and are not due to pressure at birth. Schleich attributes them to hindrance of circulation and blood stasis due to pressure. Naumoff thinks that there is a special interference with the retinal circulation owing to increased intra-cranial pressure. E. v. Hippel considers that the absence of dilatation of the intervaginal space is against this theory. Thomson and Buchanan attribute them to increase of blood-pressure caused by obstruction of the placental circulation, sudden relaxation of pressure during birth throwing a strain upon the relaxed vessels. Wolff considers that the most important factor is asphyxia, and there is much to be said in favour of this theory. Coburn described hæmorrhage into the iris, sclerotic and choroid, but Wintersteiner failed to find them in the ciliary body or sclerotic.

KÖNIGSTEIN.—Wiener med. Jahrbuch, 1881. SCHLEICH.—Mitth. aus d. ophth. Klinik in Tübingen, 1884. NAUMOFF.—A. f. O., xxxvi, 3, 1890. TRUC.—Ann. d'Oc., cxix, 1898. NETTLESHIP.—T. O. S., xxiii, 1903. E. v. HIPPEL.—A. f. O., xlv, 2, 1898. *THOMSON AND BUCHANAN.—T. O. S., xxiii, 1903. *WOLFF.—Hirschberg's Festschrift, Leipzig, 1905. COBURN.—A. f. A., liv, 1906. WINTERSTEINER.—Z. f. A., ii, 1899.

Formation of aneurismal dilatations.—Lionville first discovered miliary aneurisms in the brain and retina in the cadaver. Galezowski and Denissenko reported the first ophthalmoscopic observations after injury to the eyes. Magnus and Fuchs record cases of communication between retinal arteries and veins following injury. The condition must be attributed to rupture of the vessels, the resulting scar being of

insufficient strength to support the normal intra-vascular pressure. When communication between an artery and vein occurs both must have been ruptured and have healed together. The amount of dilatation is slight, owing to the support afforded by the intra-ocular pressure.

GALEZOWSKI.—Rec. d'O., 1874. DENISSENKO.—Wiener med. Presse, 1881. MAGNUS.—Virchow's Archiv, lx, 1874. FUCHS.—A. f. A., xi, 1882.

Rupture of the retina.—Rupture of the retina often occurs in cases in which the sclerotic is ruptured by a blow, though this membrane may escape even in severe cases. Injuries with shot in which the optic nerve is torn through or dragged forcibly backwards may be associated with circumpapillary rupture of the retina. It has been mentioned that retinal rupture is a relatively infrequent complication of rupture of the choroid (v. p. 1151).

Isolated rupture of the retina may also occur as the result of contusion (Dohmen, Hock, and others). Splits in the retina, complete or involving only certain layers, are rare in the posterior part of the fundus. Tears of the retina at the ora serrata occur (Scheffels, Baquis, Wintersteiner, Römer, Hesse), but these also are rare in the absence of perforating wound. Fuchs records a case occurring as the result of reclination of a shrunken cataractous lens. Oeller reports a case of macular rupture from a shot wound in the orbit. Rupture is common in detached retinæ (*vide infra*).

DOHMEN.—K. M. f. A., v, 1867. HOCK.—Bericht des k. k. allgem. Krankenhauses Wien, 1865. SCHEFFELS.—A. f. A., xxii, 1891. BAQUIS.—Ann. di Ott., xxv, 1896. WINTERSTEINER.—B. d. o. G., 1901. RÖMER.—K. M. f. A., xxxix, 1901. HESSE.—Z. f. A., xvii, 1907. FUCHS.—K. M. f. A., xv, 1877. OELLER.—Annal. des städt. allgem. Krankenhauses München, i, 1878.

" *Hole* " *at the macula.*—The appearance of a hole at the macula is usually the result of contusion of the globe. It consists in a perfectly circular red spot, one third to one half the diameter of the disc. It is situated exactly at the fovea and the surrounding retina is slightly opaque in the early stages. The first cases were described by Knapp (1869), and Noyes (1871); in 1900 Kuhnt, Haab, Treacher Collins, and Ogilvie added twenty-seven cases. The first anatomical examination was made by Pagenstecher and Genth (1875), followed by others by Fuchs, A. H. Pagenstecher, Murakami, E. v. Hippel, and Coats. The condition may certainly arise from disease without trauma. Reis elaborates the theory foreshadowed by Fuchs that retinal œdema is the underlying lesion. This view is supported by cases arising in connection with retinal vascular disease, amaurotic family idiocy (q. v.), etc. Ogilvie regards it as due to *contrecoup*, but a hole has never been observed within sixty hours of the contusion, whilst opacity of the retina has been observed in cases which subsequently developed a hole (Reis, Holden, Kuhnt, de Schweinitz). No case has been seen ophthalmoscopically and examined microscopically, if my case of amaurotic family idiocy be excluded. In several cases there is a history of iridocyclitis (*cf.* Fuchs, E. v. Hippel, de Schweinitz). Coats's views are summarised as follows : Macular holes are produced by an œdema

FIG. 792.—" HOLE " AT MACULA.

Ogilvie, T. O. S., xx.   From a boy, æt. 12, who was struck under the left eye with a stone nine months previously.
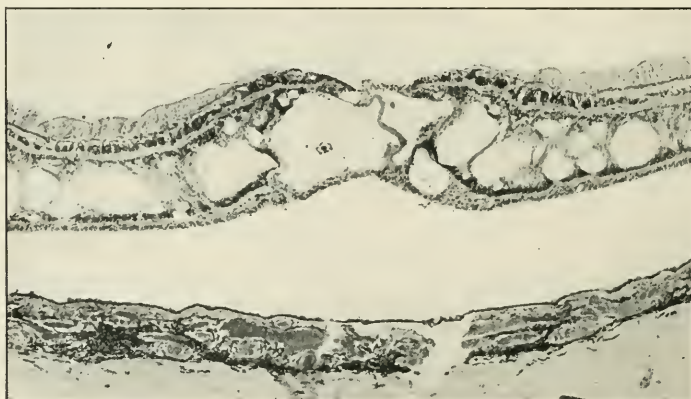
FIG. 793.—CYSTIC DEGENERATION AT MACULA.   × 32.

Coats, R. L. O. H. Rep., xvii.   Cystic degeneration at the macula, a preliminary stage to the development of a hole.

of the retina at the posterior pole. The œdema may not be confined to the macular region, but the appearance of a hole will be produced only if there is a defect at least of the inner layers of the retina. Possibly for a completely typical picture without membranes or shreds a total defect of all the layers is necessary, and it has been proved that such a defect can arise from retinal œdema (Coats). The œdema may result from contusion (commotio retinæ), from toxins due to

FIG. 794.—HOLE AT MACULA. × 75.
Coats, R. L. O. H. Rep., xvii. Hole at the fovea centralis, from an eye which had been injured a month previously by a fall.

iridocyclitis, or from retinal vascular disease. Rupture of the retina at the time of injury is not the cause of macular holes.

KNAPP.—A. f. A., i, 1869. NOYES.—T. Am. O. S., 1871. PAGENSTECHER AND GENTH.—Atlas, pl. xxv, figs. 1 and 4, 1875. HOFFMANN.—K. M. f. A., xxiii, 1885. HARTRIDGE.—T. O. S., ix, 1889. LORING.—Text-book of Ophthalmoscopy, ii, 1891. LAWFORD.—T. O. S., xiii, 1893. KUHNT.—Z. f. A., iii, 1900. HAAB.—Z. f. A., iii, 1900; Atlas, 1900; B. z. A., l, 1900. TREACHER COLLINS.—T. O. S., xx, 1900. *OGILVIE.—T. O. S., xx, 1900. FUCHS.—Z. f. A., vi, 1901. HARMAN.—T. O. S., xxi, 1901. A. H. PAGENSTECHER.—A. f. O., lv, 1902. MURAKAMI.—A. f. O., liii, 1902. WARD HOLDEN.—New York Acad. of Med., 1903. DE SCHWEINITZ.—T. Am. O. S., x, 1904. WISSELINK.—K. M. f. A., xliii, 1905. E. v. HIPPEL.—A. f. O., lxiv, 1906. REIS.—Z. f. A., xv, 1906. QUINT, KÜSEL.—K. M. f. A., xliv, 1906. POYNTON, PARSONS, AND HOLMES.—Brain, 1906. *COATS.—R. L. O. H. Rep., xvii, 1, 1907.

Detachment of the retina *(ablatio retinæ, amotio retinæ).*—The retina is probably usually detached in cases of injury with a blunt object causing rupture of the globe. It is not very common in cases in which the globe is not ruptured; many of these are "idiopathic," *i. e.* the cause is unknown, and the history which is often elicited after the event of slight injuries has no ætiological importance. Two types of traumatic detachment must be distinguished: (1) those with hæmorrhage beneath the retina; (2) those with albuminous fluid. In

the former case the detachment is likely to be shallow and the retina ruptured ; most frequently both choroid and retina are ruptured (*cf.* p. 1151). Although this is rare detachment by hæmorrhage without rupture is still rarer (Praun). Cases are recorded by Cooper, Hoering, Hock, Schmidt-Rimpler.

Detachment of the retina with the accumulation of serous fluid beneath it has not yet been satisfactorily explained. It is most likely to occur in highly myopic eyes, which are predisposed to it (*v.* Vol. III, p. 920). In most cases—according to some investigators in all—the retina is ruptured (Nordenson), and fluid passes through the rent from the vitreous. It is certain that in many cases no rupture of the retina can be seen ophthalmoscopically, and it is probable that it is not an indispensable factor. If so, the fluid must be derived from the choroid, or from the vitreous by a process of osmosis. Such experiments as have been made on the latter possibility have not been conclusive.

The possibility of reattachment of the retina is greatest in the traumatic cases. Müglich has collected 136 such cases from the literature—42 times in myopic eyes, 11 with retinitis albuminurica, 12 times after trauma, 10 times after syphilitic choroiditis, 5 times after other forms of choroiditis, 3 times after operation, 3 times with inflammatory processes in the neighbourhood of the eye, and 50 times in cases of unknown origin. Too much stress must not be laid upon these statistics. Adamük has reported three cases of reattachment after trauma.

Almost invariably the tension is diminished, but Hoor reports a case with increased tension. It is probable from the account of this case that the glaucoma was independent of the detachment.

Detachment of the retina is not uncommon following shot wounds in the orbit. They may be due to hæmorrhage (Höring) or to sub-retinal serous exudation. In most cases the globe is ruptured, but detachment may occur without direct injury to the eyeball.

v. GRAEFE.—A. f. O., i, 1, 1854. H. MÜLLER.—A. f. O., iv, 1, 1858. IWANOFF.—A. f. O., xv, 2, 1869. HÖRING.—K. M. f. A., ix, 1871. GENTH.—K. M. f. A., x, 1872. GOLDZIEHER.—A. f. O., xix, 3, 1873. SANITÄTSBERICHT ü. d. deutschen Heere im Kriege gegen Frankreich 1870–71, iii, Berlin, 1888. v. OETTINGEN.—Die indirecten Läsionen des Auges, Stuttgart, 1879. SCHWEIGGER.—A. f. A., xii, 1883. NORDENSON.—Die Netzhautablösung, Wiesbaden, 1887. RAEHLMANN.—A. f. A., xxvii. 1893. FALCHI.—A. f. O., xli, 1895. NUEL.—A. d'O., xvi, 1896. HEINE.—A. f. A., xxxviii, 1899. PICHLER.—Z. f. A., iii, 1900. VELHAGEN.—A. f. O., xlix, 1900. AXENFELD AND YAMASHITA.—B. d. o. G., 1900. LAWFORD.—T. O. S., xxi, 1901. PFALZ.—Z. f. A., x, 1903. GONIN.—Ann. d'Oc., cxxxii, 1904. LEBER.—K. M. f. A., xlii, 1904. PAUL.—K. M. f. A., xliii, 1905. BEST.—K. M. f. A., xlii, 1904 ; B. d. o. G., 1906. DOR.—Ann. d'Oc., cxxxviii, 1907.

White streaks often appear in the retina after detachment, especially in those cases in which partial or complete reposition occurs. They are usually for the most part radial to the disc (Onisi, Caspar), but many show a radial disposition around some other spot in the fundus (Haab), and irregular streaks are common. Sometimes they are arranged with great regularity parallel to each other (Nettleship, Praun), in which case they generally run more or less horizontally. These streaks are due to organisation of the subretinal exudates, which thus tie the retina down again to the choroid. The process must

commence at the margin of the detachment or at sites where contact of the folds with the choroid occurs. The streaks may be pigmented at the edges or indiscriminately (Nettleship, Praun), but they are often quite white. The presence of pigmentation is no evidence of previous choroido-retinitis (Praun). The direction of the striæ depends chiefly upon the directions of greatest tension in the retina, since these naturally determine the disposition of the folds.

Other streaks occur in relationship with the vessels; these may be independent of hæmorrhages but are frequently associated with this cause. Prevascular striæ may show all stages towards the development of "retinitis proliferans" (q. v.). Perivascular striæ are associated with degeneration of the walls of the vessels, and are usually due to general disease, such as Bright's disease—which has indeed been found in a considerable number of "idiopathic" cases of detachment of the retina, or in cases of detachment following albuminuric retinitis; occasionally the vascular degeneration appears to be purely local, affecting only the vessels of the detached area. Organisation of the subretinal exudates may show a special tendency to be distributed in the neighbourhood of the vessels, forming one group of retrovascular striæ (Jaeger, Liebreich, Zehender, de Wecker, Onisi, Nagel, Nettleship, Caspar, Haab, Goerlitz, Praun). Retrovascular striæ also occur as the result of parenchymatous retinal hæmorrhages, especially in cases of general arterio-sclerosis. They are usually pigmented branching streaks, showing an astonishing resemblance in distribution to blood-vessels—*angioid streaks* (de Schweinitz, Doyne, Fretori, Frost, Plange, Stephenson, Knapp, Walser, Holden, Lister).

All the above types of retinal striation must be carefully distinguished from the streaks due to sclerosis in a retina which has been detached for a long period. These consist of bands of fibrous tissue in the retina, showing a predilection for the neighbourhood of the larger vessels (*e.g.* de Wecker, Landolt).

LEBER.—In G.-S., v, 1877. MÜGLICH.—Dissertation, Marburg, 1891. ADAMÜK.—Ophth. Bote, Kiew, 1890. HOOR.—Wiener klin. Woch., 1888. COOPER.—Wounds and Injuries of the Eye, London, 1859. HÖRING.—K. M. f. A., ix, 1871. HOCK.—Wiener med. Presse, 1880. SCHMIDT-RIMPLER.—A. f. A., xii, 1883. CASPAR.—A. f. A., xxx, 1895. NETTLESHIP.—T. O. S., iv, 1884. ONISI.—Dissertation, Tübingen, 1890. PRAUN.—B. z. A., xii, 1895; Die Verletzungen des Auges, Wiesbaden, 1899. BANHOLZER.—A. f. A., xxv, 1892. GOERLITZ.—K. M. f. A., xxxv, 1897. DE SCHWEINITZ.—T. Am. O. S., 1896. WALSER.—A. of O., xxv, 1896. DE WECKER AND LANDOLT.—Traité, iv. KRÖNER.—A. f. A., lvi, 1906. DOYNE.—T. O. S., ix, 1889. FRETORI.—B. z. A., xxiv, 1898. FROST.—The Fundus Oculi, Edinburgh, 1896. HOLDEN.—A. of O., xxiv, 1895. PLANGE, KNAPP.—A. of O., xxi, 1892. STEPHENSON.—T. O. S., xii, 1892. LISTER.—Ophth. Rev., xxii, 1903.

III. PENETRATING INJURIES WITHOUT RETENTION OF A FOREIGN BODY.

Wounds of various parts of the eye have already been described in the first and second volumes of this work.

IV. PENETRATING INJURIES WITH RETENTION OF A FOREIGN BODY.

Cornea.—Foreign bodies in the cornea are extremely common and of the greatest variety. They may be superficial or deep, wounding

the epithelium alone or both epithelium and substantia propria.   As a
rule they irritate the eye only mechanically, and the greatest danger
associated with them is due to the pathogenic organisms which they
carry in with them or allow to enter by breaking down the epithelium.
Non-septic foreign bodies cause wounds which heal rapidly when the
source of irritation is removed.   Septic foreign bodies cause wounds
which give rise to ulcers, thus often loosening the foreign body and
leading to its extrusion.   The resulting ulcer behaves variously
according to the nature of the organisms present.

   Some foreign bodies cause chemical irritation owing to the action
of the ocular fluids upon them.   Thus particles of iron rapidly give
rise to a ring of rusty staining around them.   According to Leber the
carbonic oxide in the fluids combines with the iron to form an acid
carbonate, which passes into oxide of iron by further oxidation.   These

Fig. 795.—Implantation Cyst of Conjunctiva.
Mayou, R. L. O. H. Rep., xvi.   A. Epithelium.   B. Wound through which the
piece of wood entered.   C. Epithelium lining the cyst.   D. Piece of wood surrounded
by leucocytes.   E. New formed vessel near the cyst.

products are not very irritating.   Cases of prolonged retention of a
foreign body without irritation are reported—glass for $2\frac{1}{4}$ years
(Magnus), iron for 2 years (Knapp), copper for 20 months (Shaw
Bowen), etc.

   Particles of stone are shown by statistics to be very prone to give
rise to hypopyon ulcer (cf. Hillemanns), possibly because they are not
generally sterilised by heat as with particles of steel.

   Caterpillar hairs may cause ophthalmia nodosa (q. v.) : cases in
which the irritation was limited to the cornea are recorded by Sedan,
Elschnig, and Natanson.   Batten has described a parasitic crustacean
(Caligus curtus) as a foreign body in the cornea.

Leber.—Die Entstehung der Entzündung, Leipzig, 1891.   Gruber.—A. f. O., xl, 2,
1894.   Knapp.—A. f. A., xii, 1883.   Shaw Bowen.—A. f. A., xvii, 1887.   Hillemanns.—
A. f. A., xxxi, 1895 ; xxxii, 1896.   Sedan.—Rec. d'O., v, 1884.   Elschnig.—K. M. f. A.,
xxxiii, 1895.   Natanson.—K. M. f. A., xxxv, 1897.   Batten.—Lancet, 1900.

Sclerotic. — Foreign bodies in the sclerotic are about 200 times less frequent than in the cornea (Praun), chiefly due to its greater protection and smaller curvature. The eye is less irritated than when the particle is retained in the cornea. Cases of prolonged retention without irritation are recorded by Raynaut (14 years), Roulet (7 years), Strawbridge (21 years), Oeller (17½ years), Treacher Collins (26 years). Foreign bodies may penetrate the globe and become embedded in the sclerotic on the opposite side.

BERLIN.—K. M. f. A., xix, 1881. HÖHNE.—K. M. f. A., xxxiv, 1896. SILEX.—Berl. klin. Woch., 1888. RAYNAUT.—In Nagel's Jahresbericht, 1887. ROULET.—Korrespondenzbl. d. Schweitzer Aerzte, 1877. STRAWBRIDGE.—T. Am. O. S., 1875. OELLER.—C. f. A., vi, 1882.—HIRSCHBERG.—A. f. O., xxxvi, 3. TREACHER COLLINS.—Ophth. Rev., xi, 1892.

Anterior chamber and iris. — Particles of iron or steel are the most common foreign bodies in the anterior chamber, and, indeed, within the eye: stone, copper (usually caps), glass, wood, etc., occur less frequently. Usually the globe is perforated through the cornea, less often through the sclerotic. They are generally caught and entangled in the iris unless they perforate the lens capsule or pass through into the vitreous. Foreign bodies in the posterior chamber seldom occur except when they are drawn forward in extracting magnetisable bodies from the vitreous. Franke has collected the cases of foreign body in the anterior chamber published up to 1884, Blessig those up to 1890.

The points of greatest pathological interest are the occurrence of sepsis and the different behaviour of various substances in producing irritation, usually due to the chemical action of the intra-ocular fluids on the foreign body. *Gold* may long remain in the eye without producing irritation, as shown by the use of gold wire for drainage (de Wecker). Cases are recorded by Stellwag and Wardrop. *Silver* acts similarly (Kipp). *Glass* may long remain without serious result, even when the lens is wounded (Ferguson), or the particle is free in the anterior chamber (Critchett). Haziness of the cornea may result from contact with its posterior surface (Höring, Wagenmann) or from chemical action (Leber). *Porcelain* acts similarly to glass.

Particles of *stone* are more liable to set up severe inflammation, often ending in suppuration, less frequently in chronic plastic irido-cyclitis. In one case the eye remained quiet for thirty-two years with a particle of stone in the iris (Riecke). Chemical irritation depends upon the composition of the stone, those containing calcium being most soluble. Delayed inflammatory reaction (Saemisch, Yvert) is generally due to fresh injury, *e. g.* contusion (Landmann). Praun attributes the greater frequency of suppuration with stone as compared with glass to the greater laceration of the wound, infection being derived from the conjunctival sac, owing especially to lacrymal complications. Cirincione has investigated the subject experimentally.

Splinters of *wood* act like stone. *Gunpowder* and chips of *shot* seem relatively non-irritating (Cooper, Kipp, Salomon, Spencer Watson, and others). The lead of shot is rapidly covered with a layer of insoluble carbonate, which prevents diffusion.

The cases of *iron and steel* on record are very numerous. The

immediate effects are usually comparatively slight, the particle remaining free in the anterior chamber or entangled in the iris, the reaction being greater in the latter event, ending in encapsulation. Suppuration is relatively uncommon, since the particles are generally aseptic and there is less laceration of the parts than with other foreign bodies. On the other hand, if the foreign body is left inflammatory reaction is almost invariably the ultimate result (see " Siderosis bulbi ").

Particles of *copper*, usually fragments of percussion caps, occur less frequently in the eye than iron or steel, but always set up severe iridocyclitis and leucocytosis. The pus formation appears to be independent of pyogenic organisms in most cases, and is a pure chemico-physiological reaction (Leber). This is further shown by the fact that the inflammation tends to cause phthisis bulbi and not perforation of the globe as in ordinary panophthalmitis: the pus formation retrogrades after a certain time and is not progressive. Rare cases of copper in the eye without severe reaction are on record (Yvert, Jaeger, Hotz, Fränkel): in those of long duration it is probably accounted for by rapid encapsulation.

Gold.—STELLWAG, WARDROP.—In Landmann, A. f. O., xxviii, 2, 1882.

Silver.—KIPP.—Am. jl. of O., 1884.

Glass and porcelain.—*FRANKE.—A. f. O., xxx, 1, 1884 (Bibliography). HIRSCHBERG.—Berl. klin. Woch., 1874. BICKERTON.—Brit. Med. Jl., 1888. FERGUSON.—Ophth. Rev., iv, 1885. WAGENMANN.—A. f. O., xl, 5, 1894.

Stone.—FRANKE.—*Loc. cit.* *PRAUN.—Die Verletzungen des Auges, Wiesbaden, 1899 (Bibliography). S. CIRINCIONE.—Z. f. A., xvii, 1907.

Wood.—FRANKE.—*Loc. cit.* PRAUN.—*Loc. cit.*

Gunpowder and shot.—Bibliographies in FRANKE, *loc. cit.*; LANDMANN, A. f. O., xxviii, 2, 1882; ZANDER AND GEISSLER, Ueber die Verletzungen des Auges, Leipzig, 1864; PRAUN, *loc. cit.* SPENCER WATSON.—Lancet, 1872.

Iron and steel.—Bibliographies in FRANKE, *loc. cit.*; PRAUN, *loc. cit.* HIRSCHBERG.—Der Electromagnet in der Augenheilkunde, 1896.

Copper.—Bibliographies in FRANKE, *loc. cit.*; PRAUN, *loc. cit.* PLITT.—K. M. f. A., xliv, 1906.

Many cases of cilia in the anterior chamber have been reported. Müller found 5 cases in 2 years in Fuchs's clinic among 30,000 patients. Vieweger collected 29 cases up to 1883; in 13 cases 1 hair, in 3 cases 2 hairs, in 2 cases 4, in 1 case 5, in 2 cases 6, in 3 cases several, in 1 case 14. In 25 cases there had been a perforating wound, by wood in 13, iron in 8, stone in 2. The wound was generally in the lower part of the cornea at some distance from the limbus. The condition may follow operation wounds. The hairs were free in the anterior chamber in 9 cases, adherent to the posterior surface of the cornea in 3, on the iris in 5, on the lens capsule in 3, in the posterior chamber in 2. Of 17 cases in which the hairs were allowed to remain the eye remained quiet in 8, in 2 sympathetic ophthalmia followed (Cunier, v. Graefe), and in 7 cysts of the iris occurred (see Vol. I, p. 315).

The hairs are usually in the lower part of the anterior chamber, often having one end embedded in the corneal scar. Cyst formation is relatively uncommon, little reaction being usually set up even after prolonged periods (Schwarz 8 years, Pagenstecher 10, Leviste 12, Meyer 13, Müller 24). Samelsohn considers that they may become

absorbed, but this view has not received confirmation. Indeed, the cilia alter scarcely appreciably in time, only the pigment being absorbed, *e. g.* in 34 years (Müller, Schwarz).

MÜLLER. — Wiener med. Woch., 1894. VIEWEGER. — Dissertation, Bonn, 1883. v. GRAEFE.—A. f. O., x, 1, 1864. SCHWARZ.—B. z. A., xxv. LEVISTE.—Ann. d'Oc., cxii, 1894. MEYER.—Wiener klin. Woch., 1894. SAMELSOHN.—K. M. f. A., xxiii, 1885. ZANDER AND GEISSLER.—Ueber die Verletzungen des Auges, Leipzig, 1864. v. ROTHMUND — K. M. f. A., x, 1872. *FRANKE.—A. f. O., xxx, 1, 1884 (Bibliography). ROBERTSON.—Fifth Internat. Congress, 1878. SCHUBERT.—Dissertation, Berlin, 1877. HOLMES.—A. f. A., xii, 1883. PESCHEL.—K. M. f. A., xxv, 1887. TREACHER COLLINS.—T. O. S., ix, 1889. v. HIPPEL.—A. f. O., xl, 1, 1894. McGILLIVRAY.—Eleventh Internat. Congress, Edinburgh, 1894. WINTERSTEINER.—A. f. O., xl, 2, 1894. FEHR.—C. f. A., xxv, 1901.

Some special results attending the presence of foreign bodies in the iris have already received attention, *e. g. ophthalmia nodosa* (Vol. I, p. 84) ; *pearl tumours and implantation cysts* (Vol. I, p. 312). Exuberance of development of granulation tissue as the result of the irritation has been described as *traumatic granuloma of the iris* (Knapp, Wicherkiewicz, de Wecker, Hirschberg and Steinheim, Alt, Berthold). The chief difficulty in these cases lies in the diagnosis from gumma, tubercle, and sarcoma. *Sarcoma of the iris* has been described as a sequel of injury by Raab, and *tubercle* by Treitel.

KNAPP.—Die intraocularen Geschwülste, 1868. WICHERKIEWICZ.—K. M. f. A., xix, 1881 ; xxxii, 1894. DE WECKER.—In G.-S., iv, 1876. HIRSCHBERG AND STEINHEIM.— A. f. A., i, 1869. ALT.—A. f. A., vi, 1877. BERTHOLD.—Berl. klin. Woch., 1877. RAAB.— K. M. f. A., xiii, 1875. TREITEL.—Berl. klin. Woch., 1885.

Ciliary body.— Foreign bodies in the ciliary body are usually due to explosions ; copper caps are commonest, sometimes iron, stone, etc. They may penetrate the sclerotic over the site or pass through the eye from the opposite side, with or without injury to the lens, or perforate the limbus or cornea and iris. They generally cause intense cyclitis, leading to shrinking of the globe, and not infrequently give rise to sympathetic ophthalmia. Doyne is of opinion that chips of china are especially liable to set up sympathetic ophthalmia.

KNAPP.—A. f. A., x, 1881. LANDMANN.—A. f. O., xxviii, 2, 1882. LEBER.—A. f. O., xxxi, 1, 1885. HIRSCHBERG.—A. f. O., xxxvi, 3, 1890. FUMAGALLI.—Ann. di Ott., xviii, 1889. DAUB.—Dissertation, Kiel, 1900.

Lens.—Common foreign bodies in the lens are iron and steel, stone, glass (Laqueur) ; less frequent wood, coal (Knabe) ; powder (Terson, Pooley, Millikin) ; shot (Pamard, Galezowski) ; bone (Spierer) : wire (St. John), etc. The lens shows great tolerance for foreign bodies, even copper, probably owing to the extremely low metabolism which induces but slight chemical change ; the foreign body may be retained as long as twenty-eight years (Treacher Collins). Traumatic cararact (q. v.) results from the opening of the capsule and entry of aqueous, and is not much influenced by the presence of the foreign body. Chemical change is not entirely absent. and staining follows in time from iron (v. Graefe, Vossius, Samelsohn, Ausin, Leber—*see* " Siderosis bulbi "), etc. Copper does not cause the intense leucocytosis which occurs when it is present in the iris, etc. (Pagenstecher, Grósz,

Hirschberg, Mendel, Wicherkiewicz, Wagenmann), and a piece of copper partly in the lens and partly protruding into the anterior chamber may cause little reaction (Leber), though in time the lens fibres take on a dirty yellow coloration. The particle may be completely extruded into the anterior chamber, in which case hypopyon will follow. Glass, stone, wood (Forlanini), etc., cause no chemical reaction.

The foreign body usually perforates the cornea, and may enter the lens in the pupillary area or perforate the iris; in the latter case the traumatic iritis causes the greatest disturbance. The aqueous may not even be lost, or is rapidly replaced. If the iris is wounded a posterior synechia forms. If the lens capsule is widely opened the swelling of the lens fibres may cause extrusion into the anterior chamber, and secondary glaucoma may ensue as in ordinary traumatic cataract. If the capsular wound is very small, especially if protected by the iris, complete opacification of the lens may be delayed, but is almost invariable. Rare cases are reported in which the cataract is limited to the track of the foreign body, or even clears up to some extent (Desmarres, Berger, Vossius, Galezowski, Snell, de Wecker, Baudry). According to v. Arlt and Becker this occurs most frequently when the periphery of the lens is involved. Rarely a foreign body may become extruded from the lens into the vitreous (iron, Landmann; glass, Post). Früchte has recently reported a case of epithelial implantation in the lens.

KNABE.—Dissertation, Halle, 1892. TERSON.—A. d'O., xii, 1892. POOLEY.—New York Med. Journ., 1871. MILLIKIN.—Ophth. Rev., xi, 1892. ST. JOHN.—T. Am. O. S., 1891. TREACHER COLLINS.—Ophth. Rev., xi, 1892. V. GRAEFE.—A. f. O., vi, 1, 1860. VOSSIUS.—K. M. f. A., xviii, 1880. SAMELSOHN.—K. M. f. A., xix, 1881. AUSIN.—Dissertation, Dorpat, 1881. BERLIN.—A. f. O., xiii, 2, 1867. LANDMANN.—A. f. O., xxviii, 2, 1882. LEBER.—Die Entstehung der Entzündung, Leipzig, 1891. FORLANINI.—Ann. di Ott., i, 1871. BECKER.—In G.-S., v, 1877. DESMARRES.—Traité, iii, Paris, 1847. BERGER. A. f. A., xvii, 1887. PAGENSTECHER.—Mitth. aus d. Augenkl. zu Wiesbaden, ii, 1862. GRÓSZ.—C. f. A., xiv, 1890. SANDFORD.—T. O. S., xiii, 1893. HIRSCHBERG.—Deutsche med. Woch., 1894. MENDEL, WICHERKIEWICZ.—C. f. A., xxii, 1898. WAGENMANN.—A. f. O., xliv, 2, 1897. LEBER.—A. f. O., xxx, 1, 1884. BARKAN.—A. f. A., iv, 1874. POST.—A. f. A., xxxiv, 1897. SCHACHLEITNER.—Dissertation, Bonn, 1881. SPIERER.—K. M. f. A., xxix, 1891. V. HIPPEL.—B. d. o. G., 1893. LAQUEUR.—A. f. A., liii, 1905. FRÜCHTE.—K. M. f. A., xliv, 1906.

Vitreous.—Foreign bodies in the vitreous are usually iron or steel, copper, stone, glass or wood; less commonly shot, powder, etc. Weidmann found that 75 per cent. were iron or steel. Copper caps are next in frequency. Particles of powder are reported by Pooley, Ballias, Bergeret, Hirschberg, and others. Rare foreign bodies are bone (Rémy), whip thong (Hutchinson), wax (Bader), cilia (de Lapersonne and Vassaux, Deutschmann, Treacher Collins, Quint). Access to the vitreous is given through the cornea, pupil and lens, through the cornea, iris and lens, through the cornea, iris and zonule, through the sclerotic; the cornea is more often perforated than the sclerotic, as with all perforating foreign bodies. Hildebrand found the four routes represented in 43 cases by 6, 16, 6, and 15 respectively. The substance may remain near the site of entry, rebound from the opposite wall, or be suspended in the vitreous: in the latter case it

eventually sinks to the bottom of the vitreous chamber owing to degenerative changes in the humour, which lead to liquefaction, partial or complete. Berlin found that in nineteen consecutive cases the body rebounded in fourteen, became embedded on the opposite side in four, and perforated the globe, passing into the orbit in one. The degenerative changes set up lead to liquefaction of the vitreous, detachment of the retina, shrinkage of the globe, etc. There is often hyphæma, rarely prolapse of iris on account of the smallness of the wound of entry, but the iris is frequently drawn over to that side. The foreign body may be visible ophthalmoscopically, and the track through the vitreous may be seen as a delicate grey line. Larger bodies cause
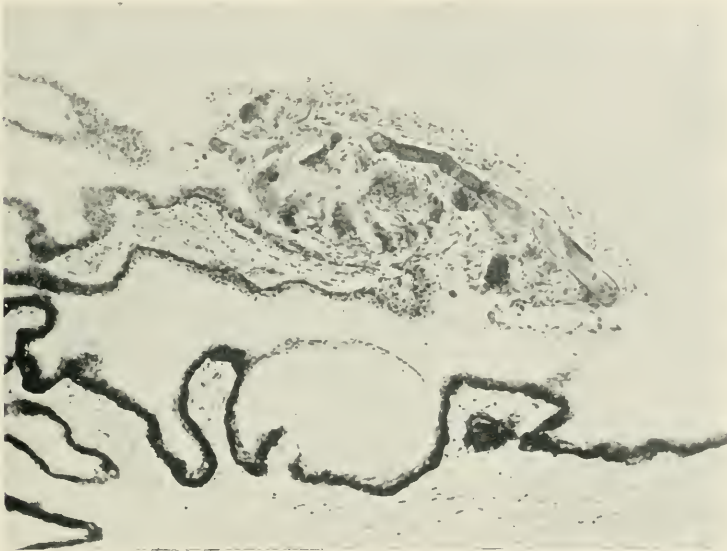
FIG. 796.—WOOD IN THE VITREOUS.
Coats, T. O. S., xxv. Showing a vegetable hair in the anterior part of the vitreous near the ciliary body. It is surrounded by loose connective tissue containing giant cells. There is a small cyst of the ciliary epithelium below.

vitreous hæmorrhage. A foreign body in the retina or choroid may be recognisable by its metallic lustre; it is usually surrounded by blood and white exudate, and in the later stages some pigmentation may occur. After prolonged sojourn it may become encapsuled. Not very infrequently air is carried in and appears as bubbles in the vitreous (Blessig, Grünthal, Morton, Herter, Hirschberg, Denti, Mittendorf, Pfalz, Meesmann, Viethaus).

The subsequent course of these cases is determined chiefly by the presence or absence of infection and of chemical action. Particles of *iron* are generally aseptic, since they are often sterilised by heat or by rapid transit through the air. In rare cases they may become encapsuled and remain indefinitely without setting up any changes

(Siegel, Landmann, Knapp, Winkler, Hosch, Meesmann, Bergmeister, Elschnig, Castelnau, Spechtenhauser, Wood). In the great majority of cases reactive organisation is abortive, so that irritation may arise from change of position at a later stage (Knapp, Mandelstamm, Landmann), and still more from chemical action (Leber, E. v. Hippel, *see* " Siderosis bulbi ").

The results of extraction of iron particles from the vitreous are of pathological interest. The immense literature which has sprung up owing to the use of giant magnets abounds in cases of remarkable cures (Hürzeler, Haab, Hirschberg, Spicer, McCallan). Unfortunately there are very few records of the late history of these cases (Praun,
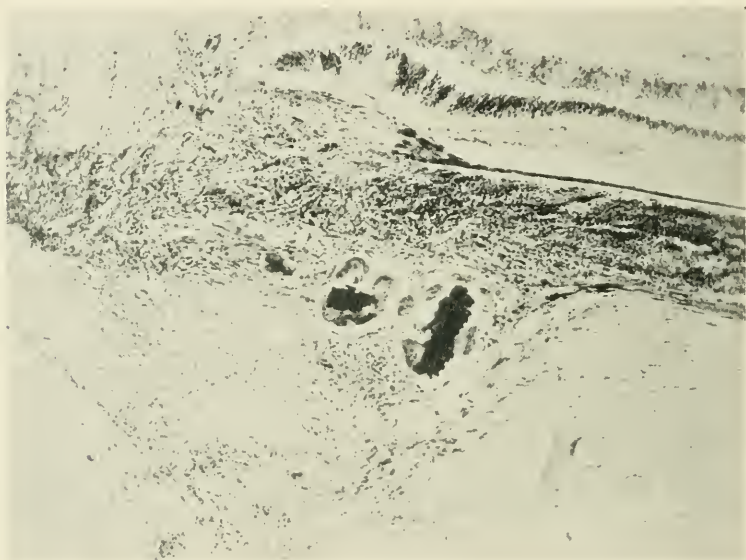
Fig. 797.—Wood in the Sclerotic.

From the same specimen, showing three pieces of bark in the sclerotic near the disc, surrounded by giant cells and inflammatory infiltration, which especially involves the choroid.

twelve years, Goulden). Certainly in a large number deterioration of vision occurs, and even loss of sight, and it is probable that this must be regarded as the usual sequel. In these cases organisation occurs along the track of entry or exit of the foreign body, and the contraction of the fibrous tissue eventuates in detachment of the retina. In other cases iridocyclitis occurs.

Copper invariably causes intense inflammatory reaction due to chemical activity, even in the absence of pathogenic organisms (Leber, Kostenitsch). There is marked leucocytosis, followed by shrinking of the vitreous, detachment of the retina, iridocyclitis, and finally phthisis bulbi. Only very rarely does a copper particle become encapsuled with restoration of useful vision (Schwarzbach, Adamük,

Weidmann, Decker). The eye may remain quiet after shrinking (Hirschberg, Ruhberg, Hoesch). Sympathetic ophthalmia is less likely to follow copper foreign bodies than others, probably owing to the intense reaction (Leber). The reaction not infrequently ends in expulsion of the body from the globe (Spectenhauser, Leber, Salzer, M. Meyer, Mason, Landesberg, Kipp, Rolland, Armagnac, Hoesch, Hoor, Denig, Raulin, Praun).

Chemical action is absent with most other foreign bodies, except *glass*, which after prolonged sojourn in the eye may lead to disorganisation of the vitreous and retina, etc. There may be little irritation for a considerable period (ten years, Grunthal), four-and-a-half months, Zirm; Maynard and Silcock). Iridocyclitis and loss of the eye must, however, be regarded as the usual sequel.

Shot and particles of gunpowder seem to become encapsuled most readily (de Wecker, v. Graefe). Stone, wood, etc., may remain for some time without setting up inflammation, which may yet follow subsequently (Praun). Stone (Cooper, Wrede), wood (Peunow), may be extruded from the eye, though less often than copper or iron.

Weidmann.—Dissertation, Zürich, 1888. Pooley.—New York Med. Journ., 1871. Ballias, Bergeret.—In Yvert, Traité, Paris, 1880. Hirschberg.—C. f. A., xiv, 1890. Rémy.—Nagel's Jahresbericht, 1874. Hutchinson.—R. L. O. H. Rep., xii, 1889. Bader. —In Zander and Geissler. de Lapersonne and Vassaux.—A. d'O., iv, 1884. Deutschmann.—B. z. A., i, 1890. Treacher Collins.—T. O. S., xiv, 1894. Burchardt.— Charité Annalen, x, 1885. Hildebrand.—A. f. A., xxiii, 1891. v. Graefe, A. f. O., iii, 2, 1857. Berlin.—A. f. O., xiii, 2, 1867; xiv, 2, 1868. Rosenmeyer.—C. f. A., xix, 1895. Leber.—Die Entstehung der Entzündung, Leipzig, 1891. Blessig.—Mitth. aus d. Petersb. Augenkl., 1894. Herter.—Nagel's Jahresbericht, 1877. Hirschberg.—Der Electromagnet in der Augenheilkunde, Leipzig, 1881. Denti.—Nagel's Jahresbericht, 1885. Pfalz.— K. M. f. A., xxv, 1887. Meesmann.—Dissertation, Berlin, 1893. Viefhaus.—Dissertation, Kiel, 1894. Siegel.—Dissertation, Tübingen, 1876. Landmann.—A. f. O., xxviii, 2, 1882. Knapp.—A. f. A., xxii, 1891; xix, 1889. Winkler.—Dissertation, Tübingen, 1886. Hosch.—A. f. A., xx, 1889. Bergmeister.—K. M. f. A., xii, 1874. Elschnig.—A. f. A., xxii, 1891. Castelnau.—In Landmann. Spechtenhauser.—Wiener klin. Woch., 1894. Mandelstamm.—K. M. f. A., xix, 1881. E. v. Hippel.—A. f. O., xlii, 4, 1896. Hürzeler.—B. z. A., xiii, 1895. Haab.—B. d. o. G., 1892. McKenzie.—R. L. O. H. Rep., xiv, 1895. McCallan.—R. L. O. H. Rep., xv, 1902. Praun.—Die Verletzungen des Auges, Wiesbaden, 1899. Kostenitsch. — A. f. O., xxxvii, 4, 1891. Schwarzbach.—A. f. A., v, 1876. Decker.—K. M. f. A., xxviii, 1890. Hirschberg. — Deutsche med. Woch., 1894. Ruhberg. — Dissertation, Kiel, 1889. Hoesch. — Dissertation, Jena, 1895. Salzer. — A. f. O., xlii, 4, 1896. M. Meyer. — Eighth Internat. Congress, Edinburgh, 1894. Mason.—R. L. O. H. Rep., ix, 1876. Landesberg. —Nagel's Jahresbericht, 1882. Kipp.—Am. Journ. of O., 1884. Rolland.—C. f. A., x, 1886. Armagnac.—A. f. A. xxxii, 1896. Hoor.—Wiener med. Woch., 1896. Denig.— K. M. f. A., xxxiv, 1896. Raulin.—A. f. A., xxxvi, 1898. Grünthal.—Berl. klin. Woch., 1895. Zirm.—K. M. f. A., xxviii, 1890. Cooper.—Injuries of the Eye, London, 1859. Wrede.—Dissertation, Bonn, 1873. Peunow.—A. f. A., xiii, 1884. Quint.—C. f. A., xxv, 1901. Coats.—T. O. S., xxv, 1905. de Schweinitz and Baer.—Am. Journ. of O., 1905. Vigier.—Ann. d'Oc., cxxxviii, 1907. Maynard and Silcock.—T. O. S., xx, 1900. Barkan.—A. of O., xxviii, 1900. Wood.—Ophth. Rec., 1900. Goulden.—R. L. O. H. Rep., xvii, 1908.

Retina.—Foreign bodies become embedded in the retina after passing through the vitreous; they may have rebounded from the fundus before reaching their final resting place. Here they may be visible ophthalmoscopically as small black specks with a metallic lustre, surrounded by a yellowish white area of œdematous retina. Later they become encapsuled in white connective tissue, with or

without deposits of black pigment. Fine pigmentary disturbance at the macula may follow (Haab), as well as extensive degenerative processes in the retina (E. v. Hippel), which often becomes detached. Encapsulation is often rapid with iron, and useful vision may persist for an indefinite time (sixteen or more years, Hirschberg). Particles more than 1—2 mm. in size are almost certain to lead to the destruction of the eye. In the absence of sepsis siderosis bulbi is the almost inevitable cause of destruction. An encapsuled foreign body may become free after a long period of quiescence. Particles of copper almost always set up suppuration in the retina and vitreous (exceptions —Priestley, Smith, Kipp [twenty-four years], Goldzieher, Hillemanns). I have pointed out that the encapsulation of foreign bodies in the retina depends largely upon their asepsis. The amount of cicatricial tissue formed in the early stages is inversely proportional to the amount of necrosis, which depends chiefly upon bacterial invasion, though mechanical injury and chemical action must also be taken into account.

Retinal degeneration in the less severe cases attacks the macula only or is generalised. In the former group yellowish white spots appear in the region of the fovea, and pigmentation may also occur. Serious disturbance of vision results, and is not recovered from (Haab, Siegfried). Generalised retinal degeneration takes the form of pigmentation resembling that of retinitis pigmentosa, and may be preceded by night blindness (E. v. Hippel). Adamük has recorded similar cases.

Apart from these causes vision or the eye may be lost from detachment of the retina, recurrent attacks of inflammation, etc., and the danger of sympathetic ophthalmia is considerable.

HIRSCHBERG.—A. f. O., xxxvi, 3, 1890; Bericht über die Augenheilanstalt Berlin, 1895. E. v. HIPPEL.—A. f. O., xlvii, 4, 1899. PRIESTLEY SMITH.—T. O. S., xii, 1892. KIPP.—Internat. Ophth. Congress, Edinburgh, 1895. GOLDZIEHER.—C. f. A., xix, 1895. HILLEMANNS.—A. f. A., xxxii, 1896. ADAMÜK.—A. f. A., xxxvi, 1898. HAAB.—Korrespondenzblatt f. Schweitzer Aerzte, xv; Atlas. *SIEGFRIED.—B. z. A., xxii, 1898. PARSONS.—R. L. O. H. Rep., xv, 3, 1903.

Choroid.—Foreign bodies, usually iron, not infrequently pass through the vitreous and become embedded in the retina or choroid. Cases are reported by Gonzenbach, v. Graefe, Schelske, and others. The behaviour is similar to that of a foreign body in the vitreous (q. v.), but encapsulation may occur, with a probability of less deleterious effect upon the eye.

GONZENBACH.—K. M. f. A., xxx, 1892. v. GRAEFE.—A. f. O., iii, 2, 1857. SCHELSKE.—Lehrbuch, Berlin, 1874.

Siderosis bulbi.—If a piece of iron is retained within the eye for a considerable time some of the metal dissolves in the intra-ocular fluids, becomes diffused throughout the eye, and stains various parts a rusty-brown colour. Attention was called to this condition by Bunge in 1890 under the designation " siderosis bulbi," but the subject had previously been investigated by Leber in 1881, in the course of his epoch-making work on inflammation. He considered that the iron

was dissolved by the carbon dioxide in the tissues, circulated as a soluble carbonate, and was deposited in insoluble form by the action of acid salts derived from the blood. Bunge divided cases of siderosis into "immediate" (unmittelbare siderosis), *i. e.* brown staining in the immediate neighbourhood of the foreign body, as is often seen in the cornea, and "remote" (mittelbare siderosis), *i. e.* staining of distant tissues from a retained particle of iron. In the latter case the iris is brown, and the cornea shows a rusty staining due to the deposit of minute granules. Still more striking in some cases is the deposit of red material in the lens, not infrequently as a circle of oval patches arranged concentrically with the margin of the dilated pupil. This staining of the lens had long been regarded clinically as pathognomonic of an intra-ocular iron foreign body. Bunge found that the staining could not be attributed to hæmorrhage. Brown granules were found in the corneal corpuscles, in the meshes of the ligamentum pectinatum iridis, on the inner surface of the ciliary muscle, and in the retina. The anterior layers of the iris were impregnated, and there were sub-capsular deposits in the lens. The retina showed complete degeneration, and Perls' micro-chemical reaction (*v.* Vol. II, p. 515) showed the whole retinal vascular system marked out by blue coloration.

The subject has been exhaustively investigated anatomically and experimentally by E. v. Hippel. In seven cases a particle of iron had been present in the eye for a period varying from ten days to seven years, five times embedded in the wall of the globe, once in the vitreous, and once in the inner surface of the ciliary body. In all cases there was marked retinal degeneration, with or without detach-ment of the retina. Perls' and other micro-chemical reactions for iron (*v.* Vol. II, p. 515) showed that certain tissues showed a specific attrac-tion for the metal. A distinction must be made between diffuse blue coloration and circumscript granular deposits which appeared brown before the application of the test. There is always intense blue coloration with the Perls' reaction immediately around the foreign body. In all cases there was diffuse coloration of the pigment epithelium of the ciliary processes and the pars ciliaris retinæ, in three cases of the capsular epithelium of the lens, in three cases of the pigment epithelium of the retina, and in three cases of the supporting tissues of the retina. The brown pigmented cells, which give a blue reaction, are found particularly in the angle of the anterior chamber and in the retina, less in the iris, and least in the choroid; they were found once in the cornea.

In four cases the foreign body passed through the globe, causing much hæmorrhage : in all of these similar changes were found. The question therefore arises as to how much of the iron reaction is directly due to the foreign body, and how much to hæmatogenous pigmentation. The coloration in the immediate vicinity of the foreign body can only be ascribed to direct solution of iron ; this may conveniently be termed "direct" siderosis. Pigmentation at a distance, "indirect" siderosis, may be either *xenogenous*, due to iron derived from the foreign body, or *hæmatogenous*, due to hæmosiderin derived from blood : both give the Perls' reaction.

Normal ocular pigment can be bleached by the prolonged action of chlorine water, which does not affect the iron pigment. Pathological iron pigment, on the other hand, is bleached in twenty-four to forty-eight hours by 5 per cent. hydrochloric acid, which does not affect the normal pigment. The retinal pigment epithelium does not normally show the iron reaction, but it possesses the power of absorbing hæmatogenous iron in a peculiar degree, so that under these conditions a blue coloration is obtained with the Perls' reaction. This capacity is not shown by the pigmented stroma cells of the choroid—a fact which has been utilised by Leber. Perls' reaction is not given with tissues which have been hardened for a prolonged period in Müller's fluid, though Quincke's method is still available.

E. v. Hippel does not consider that a true siderosis of the cornea has been proved: he thinks that the brown coloration is probably due to deposits of hæmosiderin. Xenogenous siderosis is due to solution of iron by the carbon dioxide of the tissues; the solution diffuses throughout the eye and is fixed by cells which have a specific affinity for iron. An insoluble combination with some substance in the protoplasm is formed, and this gradually becomes oxidised. In hæmotogenous siderosis the iron is already in a soluble form, and is fixed by the cells in the same manner as xenogenous iron. Greenish and greenish brown discoloration of the iris and cornea may be due to colouring matter derived from the blood. The characteristic ring of brown spots under the lens capsule (v. Graefe, Leber, Samelsohn, Landmann, Fuchs, Vossius, Ausin and others) is caused by deposition of iron in circumscribed aggregations of newly-proliferated capsular epithelial cells. Leber has shown that the introduction of a particle of iron into the vitreous causes extreme degeneration of the retina. Peculiar large granular cells are found which are derived for the most part from the retinal pigment epithelium. Injection of blood into the vitreous may cause detachment of the retina (Pröbsting), and degenerative changes are set up which resemble those due to intra-ocular iron. Injection of blood into the vitreous after puncture of the anterior chamber may cause bursting of the anterior capsule of the lens due to some unexplained mechanism.

Leber's theory of the diffusion of iron in the eye is not universally accepted. Ferrous oxide is slightly soluble in water containing a trace of oxygen (1 in 150,000). It has also been suggested that the iron is dissolved by acid phosphates in the intra-ocular fluid, or that the iron may enter into solution in organic form as an albuminate or in combination with an organic acid. The brown precipitate in the tissues is almost certainly produced by oxidation, but it is not a simple oxide or hydroxide, as it is only very slightly soluble in oxalic acid (McMullen). Probably the deposit is not identical in all cases, for different observers have recorded contradictory observations with regard to its solubility in various reagents. Ausin found the deposits in the lens insoluble in hydrochloric acid, E. v. Hippel soluble in dilute hydrochloric acid within twenty-four hours. Bunge stated that the siderotic pigment is dissolved within twenty-four hours by 5 per cent. HCl, whilst hæmatogenous pigment was scarcely altered. In

McMullen's case sections placed in 5 per cent. HCl showed scarcely any change in twenty-four hours, and only a small proportion of the brown pigment was dissolved after four days.

The oxidation of the iron compound in solution was supposed by Leber and Bunge to be effected by free oxygen derived from the arteries. Bunge found that the deposit was densest near the arteries, but other writers have not confirmed this observation. A serious objection to this simple theory is that in most cases the deposit is almost exclusively intra-cellular, and that the majority of the cells in which it is found are not phagocytic, so that they cannot be supposed to have taken up granules deposited in their neighbourhood. There is no doubt that certain cells show a special affinity for iron, and it must be concluded that precipitation occurs within the cells, a process which McMullen compares with metallic impregnation methods of staining, though this is certainly due to reduction, not oxidation.

It is probable that E. v. Hippel lays too much stress upon the possibility of hæmatogenous pigmentation in these eyes. In many cases the amount of hæmorrhage which has taken place is remarkably slight. The amount of available iron from this source is small compared with that from the foreign body, and it is likely that almost all of the staining is xenogenous, including that of the cornea.

HIRSCHBERG.—A. f. O., xxxvi, 3, 1890. LANDMANN.—A. f. O., xxviii, 2, 1882. VOSSIUS.—A. f. O., xxxi, 2, 1885 ; xxxv, 2, 1889 ; Deutsche med. Woch., 1891, B. d. o. G., 1901. v. GRAEFE.—A. f. O., vi, 1, 1860. SAMELSOHN.—K. M. f. A., xix, 1881. BUNGE.—Internat. Congress, Berlin, 1890. LEBER.—Internat. Congress, London, 1881 ; Die Enstehung der Entzündung, Leipzig, 1891. AUSIN.—Dissertation, Dorpat, 1891. *E. v. HIPPEL.—A. f. O., xl, 1, 1894; xlii, 4, 1896. HERTEL.—A. f. O., xliv, 2, 1897. SATTLER.—Internat. Congress, Utrecht, 1899. EISENBERG.—Dissertation, Giessen, 1901. CRAMER.—K. M. f. A., xl, 1902. UHTHOFF.—Deutsche med. Woch., 1903. BARKAN.—Ophth. Rec., 1903. BASSO.—Ann. di Ott., xxxii, 1903. ANDRESEN.—Dissertation, Giessen, 1903. NATANSON.—B. d. o. G., 1903. SCHULEK.—Z. f. A., xiv, 1905. PIHL.—A. f. O., lx, 1905. ROGMAN.—Ann. d'Oc., cxxxiii, 1905. WAGENMANN.—Münchener med. Woch., 1905. KIPP.—T. Am. O. S., 1906. GRÄFENBERG.—A. f. A., lv, 1906. HORN.—Dissertation, Giessen, 1906. McMULLEN.—Private communication.

V. DISPLACEMENT OF THE EYE AS A WHOLE.

Displacements of the eye as a whole are described variously as luxatio, dislocatio, and avulsio bulbi respectively. It is convenient to restrict the term "luxatio bulbi" to displacement outside the lids, "avulsio bulbi" to almost or quite complete severance of the eyeball from the body, and "dislocatio bulbi" to displacement into one of the cavities bounding the orbit (Birch-Hirschfeld). Luxation and avulsion of the globe may be considered together since no sharp limitation can be drawn between them.

Luxatio and avulsio bulbi may be divided into three groups : (1) birth injury ; (2) self-inflicted injury ; (3) accident. In all cases the displacement is facilitated by relative diminution in the orbital space, which may be due to deformity of the skull (Donaldson, Uhthoff), or any cause which renders the eyeball unduly prominent. Luxation may occur spontaneously or be brought about at will in some people

with prominent eyes and lax fasciæ (Willemer, Lewin, Birch-Hirschfeld). It is rare in the case of proptosis due to orbital tumours, etc., probably owing to the normal resistance of the fasciæ and to abnormal hypertrophy and adhesions.

Birch-Hirschfeld has collected eighteen cases due to injury during parturition. In several cases reported by Wolff, Servel, Reese, Rouchut, Tarpet, and Thomson and Buchanan exophthalmos was present, but the globe did not protrude in front of the lids. In fifteen cases forceps were used, often when the head was still high up in the pelvis. In most cases there was a narrow pelvis, and there is no doubt that this alone may be a sufficient cause (Hofmann, Sidler-Huguenin). It practically never occurs with an after-coming head. It may be due to the finger of the accoucheur (de Wecker, Beck). In many cases orbital fracture has been caused by the forceps (Maygrier, Zangerol, Coccius, Gad), and the mechanism of the injury seems to be in the first place the forcing forwards of the globe by the diminution of the orbital space with the forceps, and secondly the arrest of the protruded globe upon the unduly exaggerated promontory of the contracted pelvis. It is improbable that the blade of the forceps alone (Beaumont) can effect the injury. The shape of the infantile orbit is an important factor (Birch-Hirschfeld).

Avulsio bulbi due to this cause shows the double action of pressure from behind effecting the protrusion and traction from in front tearing the eyeball out. The muscles separate near the tendinous insertions into the globe, the external rectus most frequently escaping (Thomson and Buchanan, Wicherkiewicz, Gad), though there are exceptions (Hofmann, inferior rectus; Bock, obliques). The optic nerve ruptures a short distance behind the globe (1 in. Snell; 18 mm. Wicherkiewicz, ½ in. Gad).

DONALDSON.—T. O. S., xxiii, 1903. UHTHOFF.—K. M. f. A., xliii, 1905. LEWIN.—Berl. klin. Woch., 1905. HOFMANN.—Monatsschrift f. Geburtshilfe, iv, 1854. GUÉNIOT.—Rec. d'O., 1875. STEINHEIM.—C. f. A., iii, 1879; Deutsche med. Woch., 1883. BERLIN.—In G.-S., vi, 1880. DITTRICH.—Wiener klin. Woch., 1892. MAGRIER.—Leçons de Clinique obstét., 1893. DE WECKER.—Ann. d'Oc., cxvi, 1896. BECK.—C. f. A., xxvi, 1902. BEAUMONT, SNELL, THOMSON, AND BUCHANAN.—T. O. S., xxiii, 1903. *WOLFF.—Hirschberg's Festschrift, Leipzig, 1905. GAD.—Ophth. Rev., xxv, 1906. *BIRCH-HIRSCH-FELD.—In G.-S., ix, 1907 (Bibliography). *BERGER AND LOEWY.—Ueber Augenerkrankungen sexuellen Ursprunges bei Frauen, Wiesbaden, 1906.

The second group of cases of luxatio and avulsio bulbi includes those due to self-inflicted injury by lunatics. Birch-Hirschfeld has collected eleven cases from the literature, but these probably give no idea of relative frequency. In five there was luxation, in six avulsion. In Dehn's case the right eye was torn out, the left hung by the optic nerve; in Ideler's there was right avulsion, left exophthalmos. The mechanism, as in birth injury, is twofold—protrusion effected by the finger pushed back into the orbit beside the globe, and traction by the hand. Berlin, Dehn, and Rothenpieler deny the possibility of complete avulsion by this means, but Axenfeld has proved it by experiments on the cadaver, though much force is required. The muscles are torn at variable distances from the globe, the optic nerve usually 2—3 cm.

behind it. There is often severe injury to the eye—rupture (Dehn, Axenfeld), retinal hæmorrhage (Cronigneau). Panophthalmitis, etc., may follow this as in birth injury (Chalupecky, Axenfeld). Reposition with restoration of sight occurred in Cooper's case. The injury may be effected in about one minute.

BERGMANN.—Allg. Z. f. Psychiatrie, iii, 1846. COOPER.—Wounds and Injuries of the Eye, London, 1859. IDELER.—Allg. Z. f. Psychiatrie, xxvii, 1871. CRONIGNEAU.—Jl. de Méd. de Paris, 1887. DESPAGNET.—Soc. d'O. de Paris, 1892. DEHN.—A. f. O., xl, 2, 1894. CHALUPECKY.—Wiener klin. Rundschau, 1895. AXENFELD.—Z. f. A., i, 1899. *ROTHEN-PIELER.—B. z. A., xxi, 1889 (Bibliography). *BIRCH-HIRSCHFELD.—In G.-S., ix, 1907 (Bibliography). NOYES.—Ophth. Rec., 1907.

Of the third group of luxation or avulsion of the globe Birch-Hirschfeld has collected seventeen cases. In fourteen there was luxation, in three only avulsion (Verhaege, Herrgott, Arcoleo): the latter were in all cases due to fall on the projecting ring of a key protruding from the lock of a door. Another case must be added in which a pugilist tore out his eye and threw it upon the ground after it had been luxated by his opponent (de Wecker). These cases strikingly confirm the mechanism of avulsion as also exemplified in the other groups, viz. protrusion by pressure from behind followed by traction from in front. The same mechanism is the cause in most cases of luxation, though the second factor may be in abeyance if the lids are unduly extensile. Usually a foreign body of peculiar shape has penetrated into the orbit—tusk of a wild boar (Flarer), cow's horn (Dzialowski), hook (Jameson), knob of a fender (Lawford): probably the action is always somewhat like a fish-hook, so that traction is exerted after protrusion has been brought about.

FLARER.—Ann. d'Oc., xix, 1848. VERHAEGE.—Ann d'Oc., xxvi, 1851. JOBERT.—Ann. d'Oc., xxvii, 1852. JAMESON.—Ann. d'Oc., xxix, 1853. HERRGOTT.—Ann. d'Oc., lix, 1867. ARCOLEO.—Giorn. d'Oftalm., 1870. RAMPOLDI.—Ann. di Ott., xvi, 1887. DE WECKER.—Rec. d'O., 1890. SULLIVAN.—New York Med. Record, xxxii, 1892. *ROTHENPIELER.—B. z. A., xxxi, 1899; xlvii, 1902. KROHN.—Dissertation, München, 1899. BALDWIN, LAWFORD.—T. O. S., xxiii, 1903. *BIRCH-HIRSCHFELD.—In G.-S., ix, 1907 (Bibliography).

Dislocatio bulbi or dislocation of the globe into one of the neighbouring cavities—antrum of Highmore, ethmoid, etc.—is always the result of very severe injury. Fractures of the floor of the orbit leading to depression are not extraordinarily rare ; as a rule they cause only traumatic enophthalmos (q. v.), so that dislocation of the eye into the maxillary antrum may be regarded as an extreme form of this condition. The earliest case of dislocatio bulbi is by Henricus Smetius a Leda (1575); the eye was displaced into the nose, and some vision is said to have been retained through the nasal aperture. Dislocation into the antrum is recorded by Becker (by a cow's horn), v. Langenbeck (fall between railway engine and tender), and Tweedy (by a cow's horn). In v. Langenbeck's case vision was normal after reposition, but a corneal ulcer developed and the eye shrank. In Tweedy's case the eye was blind though there was a red reflex.

SMETIUS.—In Waltz, C. f. A., xxiii, 1899. BECKER.—A. f. O., xii, 2, 1866. v. LANGENBECK.—A. f. O., xiii, 2, 1867. TWEEDY.—Lancet, 1881. KALT.—Soc. d'O., 1905.

VI. Injuries of the Optic Nerve.

The optic nerve may be injured in any part of its course, viz. (1) in its intra-cranial part ; (2) within the optic foramen ; (3) in its intra-orbital part, *i. e.* between the optic foramen and the eye ; (4) in the scleral canal. Injuries affecting the intra-cranial portion belong rather to neurology than ophthalmology, and will not be discussed here.

Injury within the optic foramen is most commonly due to fracture of the base of the skull, rarely to direct wound or foreign body. Most of the latter are shot wounds (Doutrelepont, Moses, and others) or wounds with bayonet, knife, ramrod (Fischer), etc.

Fracture of the base of the skull implicating the optic foramen have been investigated by Berlin, v. Hölder, Leber, Nuhn, and others. In v. Hölder's cases two thirds were shot wounds, one third falls on the head. Nine tenths of Leber's cases were falls on the head ; others were due to blows on the head, etc. The injury may probably occur during birth from forceps pressure. Blows applied to the skull in any situation may cause the injury ; shot wounds are usually suicidal through the mouth. v. Hölder records a case in which the patient was run over, Vossius one in which the patient fell in a sitting posture. Prescott Hewett found that the fracture extended into the orbital roof in 23 cases out of 68 fractures of the base. v. Hölder in 124 cases of fracture of the skull found 86 fractures of the base with 79 of the orbital roof ; in 54 or 60 per cent. the walls of the optic foramen were broken. Bergmann showed that frontal fractures and those passing forwards or inwards in the middle fossa all tend to pass through the foramen, the latter sometimes passing through both and surrounding the clinoid processes. The injury is, however, only occasionally bilateral. Other post-mortem records are given by Koeller, Flatten, Greder, and others.

Hæmorrhage plays a large part in the injury of the nerve. v. Hölder, in fifty-four fractures found hæmorrhage into the nerve sheath in forty-two, and never without fracture through the canal. The blood may come from the cranial cavity, from the vessels of the sheath, or from the central vessels before they enter the nerve (Berlin). Most cases belong to the first group ; those of the second show slighter but progressive effects. According to Berlin and v. Hölder the thin ophthalmic artery, with a lumen of only 0·75 mm., is only rarely ruptured in its course through the foramen. Kuhnt draws attention to a central posterior vein in the nerve which may cause bleeding into the substance of the nerve. Pressure of blood in the intervaginal space doubtless causes disturbance of vision either by direct pressure on the nerve-fibres or by impeding the circulation (J. Meyer, Knapp, Münchow).

The nerve itself is often torn, partially or completely (v. Hölder), or small hæmorrhages are found in its substance (Demme). This fact is not surprising when the intimate union between the dura and the periosteum in this situation is considered. Probably splinters of bone, especially from the clinoid processes or roof of the orbit, may also cause severance. In Berlin's cases there was amaurosis or a high

degree of amblyopia in twenty-seven, permanent blindness occurring in twenty-four. In many cases, *e. g.* Leber, the field of vision showed localised destruction of the nerve.

If the nerve is completely severed there is blindness with a normal fundus in the early stages. In three or four weeks signs of optic atrophy appear and progress to total atrophy. The first signs of atrophy may be visible in fourteen days, in other cases it is delayed to twenty-six or more days (Leber). The pathology of this retrograde distal degeneration of the optic nerve has already been discussed (Vol. II, p. 687). Diminution in size of the retinal vessels in the early stage points to bleeding into the sheath, and may be accompanied by hæmorrhages on the disc and retina.

These may cause pigmentation of the disc (Liebreich, Hutchinson, Reich, Leber, Berlin, Knapp). Slow hæmorrhage is indicated by progressive contraction of the field of vision, which may improve at a later stage. Cases of atrophy following severance of the nerve are reported by Leber and Deutschmann, Galezowski, Schmidt-Rimpler, Debièrre, Köhler, Natanson, Münchow, and others; of early retinal anæmia by Vieusse, Nieden, Nettleship, and others; of pigmentation in the fundus by Chauvel, Yvert, and in the Sanitätsbericht; of partial injury and atrophy by Leber, Baer, Seggel, Münchow; of blood in the sheath by Meyer, Samelsohn, Roosa and Ely, Beck, Vossius, Hirschberg, Schweiger, Seggel, Bruhn, Baer, Gengnagel, Nicolini, and others; of atrophy as a birth injury by Truc, de Beck, Sidler-Huguenin, Koppen (*see* Wolff).

Doutrelepont.—Deutsche Z. f. Chir., xviii. Moses.—Dissertation, Würzburg, 1886. Fischer.—Deutsche Z. f. Chir., xviii. Hutchinson.—R. L. O. H. Rep., v, 1866; vi, 1869; vii, 1871. *Berlin, v. Hölder.—In G.-S., vi, 1880. Leber.—In G.-S., v, 1877. Leber and Deutschmann.—A. f. O., xxvii, 1, 1881. Nuhn.—Handb. d. chir. Anat., ii. Vossius. —K. M. f. A., xxi, 1883. Prescott Hewett.—Med.-Chir. Trans., xxxvi, 1853. Bergmann. —Die Lehre von den Kopfverletzungen, Stuttgart, 1880. Köhler.—Z. f. Chir., 1886.— Flatten.—Vierteljahrsschrift f. gerichtl. Med., 1890. Greder.—Z. f. Chir., 1885. Kuhnt. —A. f. O., xxv, 4, 1879. Knapp.—A. f. A., i, 1869; A. f. O., xiv, 1, 1868. Münchow.— Dissertation, Halle, 1892. Galezowski.—Gaz. hebd., 1880. Schmidt-Rimpler.—A. f. A., xii, 1883. Debièrre.—Jl. d'Oc., xi.—Natanson.—Petersb. med. Woch., 1889. Vieusse. —Rec. d'O., 1875. Nieden.—B. d. o. G., 1881. Nettleship.—A. f. A., xxxii, 1896. Chauvel.—Bull. de la Soc. de Chir., 1881. Yvert.— Traité, Paris, 1880. Baer.—A. f. A., xxxi, Ergänzungsheft, 1895. Seggel.—A. f. A., xxiv, 1892. Meyer, Samelsohn.—B. d. o. G., 1879. Roosa and Ely.—A. f. A., x, 1881. Beck.—Deutsche Z. f. Chir., xvi. Vossius.— K. M. f. A., xxi, 1883. Hirschberg.—C. f. A., viii, 1884. Schweigger.—A. f. A., xiii, 1884. Bruhn. — Dissertation, Kiel, 1889. Gengnagel. — Dissertation, Giessen, 1894. Nicolini.—C. f. A., vi, 1882. Truc.—Ann. d'Oc., cxix, 1898. de Beck.— France médicale, 1889. Sidler-Huguenin. — Korrespondenzbl. f. Schweizer Aerzte, 1903. Koppen.— Ophth. Klinik, 1902. Wolff.—Hirschberg's Festschrift, Leipzig, 1905. *Praun.—Die Verletzungen des Auges, Wiesbaden, 1899. Uhthoff.—B. d. o. G., 1901. Brun.—Beiträge z. klin. Chir., xxxviii, 1903. Graf.—Deutsche Z. f. Chir., lxviii, 1903. Liebrecht.— A. f. A., lv, 1906. Birch-Hirschfeld.—A. f. O., lxv, 3, 1907.

Injury of the optic nerve in the orbit between the optic foramen and the globe may involve the nerve before or after the central vessels of the retina have entered it. The signs and results are different and demand separate consideration.

Injury of the optic nerve *between the optic foramen and the point of entry of the central vessels* is not a very infrequent accident. The orbital

portion of the nerve is 28—29 mm. long, and the central vessels enter 15—20 mm. behind the globe. Not only is the distance but also the site of entry variable, the latter being usually down and out (Vossius), or down and in (Deyl). Cases of injury have been collected by Aschmann, Leber, and Schliephake, the last of whom found twenty-two undoubted cases of severance behind the entry of the central vessels. Later records of such cases have been given by Vessely, Szili, Seggel, and others. The injury is caused by a sharp instrument, stick, umbrella, etc., and the external wound is generally at or near the inner canthus (Zander and Geissler). A blow near the root of the nose is very likely to be diverted into the orbit here, whereas one at the outer canthus is often diverted towards the temporal region. The caruncle and semilunar fold are often divided, and the lacrymal sac may be wounded. The signs are similar to those of injury in the optic foramen, and consecutive atrophy follows in the same manner. Apart from direct injury to the eye there is generally no change in the fundus in the early stages.

YVERT.—Traité, Paris, 1880. VOSSIUS.—A. f. O., xxix, 4, 1883. DEYL.—Anat. Anzeiger, xi. ASCHMANN.—Dissertation, Zürich, 1884. LEBER.—In G.-S., v, 1877. TEIRLINK. —Ann. d'Oc., xiv, 1845. HÜBSCH.—Ann. d'Oc., xxx, 1854. SCHIESS-GEMUSEUS.—K. M. f. A., viii, 1870. JUST.—K. M. f. A., xi, 1873. SCHWEIGGER.—K. M. f. A., xii, 1874. EMMERT.— K. M. f. A., xiii, 1875. LAWSON.—Lancet, 1875. TREITEL.—A f. A., x, 1881. KÖHLER.— Z. f. Chir., 1886. SCOTT.—T. O. S., xix, 1899. GARRARD.—T. O. S., viii, 1888. MENDEL. —Berliner klin. Woch., 1899. CASPAR.—A. f. A., xli, 1900. PÉCHIN.—Ophth. Klinik, 1901. *BIRCH-HIRSCHFELD.—K. M. f. A., xl, 1902. LIEBRECHT.—Münchener med. Woch., 1905. v. MICHEL.—B. d. o. G., 1905. VESSELY.—Militärarzt, Wien, 1889. SZILI.—Prager med. Woch., 1893. SEGGEL.—A. f. A., xxiv, 1892.

Injury of the optic nerve *between the site of entry of the central vessels and the globe* is rare. I have published a case and collected those (about a dozen) previously recorded. The points of special interest are the appearance of the fundus shortly after the injury and the subsequent revascularisation of the retina. The picture at first resembles that of embolism of the central artery. There is very marked œdema at the posterior pole, but the cherry-red spot at the macula is seldom present. Cases of Krönlein's operation with retention of the globe are particularly suitable for observing the changes which occur.

Division of the optico-ciliary bundle experimentally was first investigated by Berlin. He operated upon rabbits, and found very extensive œdema of the whole retina. Leber confirmed the results in the cat. It is not easy to divide the central vessels in lower mammals, as they enter the eye close to the sclerotic.

The experiments were extended by Krenchel, who operated on frogs, and the whole subject was very exhaustively investigated by Wagenmann. The latter found that simple section of the nerve and central vessels did not cause retinal œdema, but this followed section of the ciliary arteries only. This observation requires confirmation. Wagenmann also attributes the pigmentation of the retina to injury of the ciliary blood supply.

A review of the cases presents a very constant group of ophthalmoscopic signs, which are of extreme diagnostic and pathological interest.

Many of the signs, such as disappearance of the vessels, temporary diminution of intra-ocular tension, etc., are only what might be expected.

The occurrence of œdema in tissues deprived of their blood supply is exemplified in many conditions in various parts of the body, and need, therefore, occasion no surprise in the retina in such conditions as embolism of the central artery or section of the optic nerve anterior to the entry of the central retinal vessels. Anæmic tissues take up
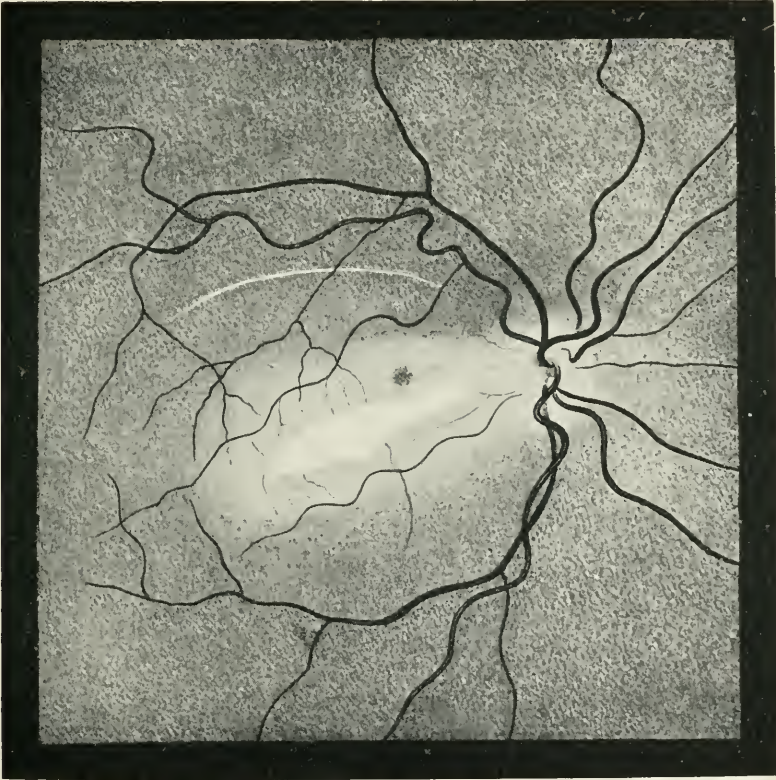

FIG. 798.—ORBITAL WOUND.
Parsons, R. L. O. H. Rep., xv.

fluid with great avidity, and this is doubtless due to the alteration in osmotic pressures brought about by the retention of katabolic products, though the exact *rationale* of the phenomenon has yet to be worked out.

It is less easy to understand why the macular region should suffer in so disproportionate a degree. The resemblance of the ophthalmoscopic picture in these wounds of the nerve to that of embolism has been pointed out by several observers. There can be little doubt that the cause is the same in both conditions. Direct injury of the posterior

pole of the eye can be definitely eliminated in several of the cases, as in my own. In the wounds of the nerve the whole retina would probably be found to be œdematous in the very earliest stage a few hours after the injury; if not, it must be due to the comparatively free vascularisation of the anterior part of the choroid by the recurrent branches of the anterior ciliary vessels. The exact parallelism between the œdema of the macular region in embolism and that in the condition under consideration is found in the fact that in the latter the macular region is the last to clear up as the retina becomes revascularised. This depends, I believe, upon the fact that, *quâ* retinal blood supply, this area is worst provided for. Hence, as the circulation is re-established, effete material will stagnate there longest, and we have seen that this is the most probable cause of the œdema.

It has already been noted by H. Pagenstecher, in his second case, that the neighbourhood of the macula seemed to be detached. I think it is probable that there was an actual slight detachment of the retina in this situation in my case. In the earliest condition the bright white summit of the area afforded no means of accurately measuring its position relative to that of the surrounding retina, which, it must be remembered, was also swollen. After the development of new vessels the difference in height was too slight to authorise a dogmatic statement. Such detachment, if present, was due to the accumulation of fluid under the retina at the most œdematous part. This may account for the absence of a cherry-red spot, but there are other reasons for this. It will be noted, in the cases previously reported, that such a spot is seldom present, though it may be simulated by a hæmorrhage, as in my case. It is mentioned in Blessig's first case, but the condition here of the vessels on the second day is scarcely consistent with division of the central trunks behind the globe.

Reported cases of embolism seem now to have proved conclusively that the red spot at the macula may be due to hæmorrhage, though in most cases hæmorrhage is absent. When it is absent we must fall back upon the theory of the contrast of the choroid, seen through the thin retina at this spot, with the surrounding œdematous, non-vascular retina. And this is a good reason why there should be no red spot when the optico-ciliary bundle is divided, even if the retina is not actually detached. Under these circumstances the choroid must be almost emptied of blood, the large veins being submitted to the intra-ocular pressure, and thus emptied. Some blood will still enter the choroid from the recurrent branches of the anterior ciliary vessels, but the choroid at the posterior pole of the eye will certainly suffer most, and we must remember that the partial anterior ciliary blood supply will prevent the intra-ocular tension from falling to zero.

No refilling of the retinal blood-vessels can occur until the cut ends of the central vessels behind the globe are sealed up. This doubtless happens quickly through retraction and clotting. As soon as organisation is sufficient to withstand the low venous pressure the vessels will commence to refill through the direct and the indirect cilio-retinal anastomoses near the optic disc.[1]   These anastomoses are rather

[1] See PARSONS, 'The Ocular Circulation,' London, 1903.

venous than arterial; hence the veins will fill up first. This is what actually occurs, and it takes place on the fourth or fifth day. The blood is, of course, derived from the recurrent branches of the intact anterior ciliary arteries, and also from any posterior ciliary arteries which may have escaped section. The retinal arteries probably fill up first from the periphery, the blood trickling in the wrong direction from the already refilled veins.

As soon as a few vessels have been refilled new capillaries will be actually created in these areas. The conditions are exactly similar to those which obtain in the vascularisation of an organising clot, etc., with the exception that the inert mass in the present instance already contains pre-formed blood channels. In both cases there is the requisite stimulus for the proliferation of the capillary walls, new endothelial tubes are formed, and these latter put on adventitial sheaths, and so anastomotic branches are formed. In this manner, by the continual opening up of the old paths and formation of new ones the circulation is re-established. The reverse current in the retinal vessels, if it occurs, is probably of short duration, the arterial anasto-moses developing rapidly under their greater internal pressure, so that a normal current is set up.

How does the blood escape from the eye? The answer to this question also explains the occurrence of venous hyperæmia and hæmorrhages. The retinal blood can no longer return by the central vein, which is now quite sealed up. It has to pass out by the same anastomoses round the disc. Here it enters the choroidal veins, is carried forward to the venæ vorticosæ, and so escapes. It is obvious that this circuitous course must offer great resistance to the exit of the blood. Further, the more blood that enters the retinal vessels, which are now, moreover, increased in number beyond the normal, the more difficult will it be for the blood to escape. It is naturally forced on by the high arterial pressure into the capillaries and veins; these become more and more congested, until finally the weaker ones give way and extravasations of blood take place.

This is the actual course of events in some of the cases, but not in all. The reason is not far to seek. The retina has ceased to be a functioning organ; it is now little more than an organising scar. The changes which occur in the new-formed blood-vessels, leading to the formation of a connective-tissue sheath, do not stop at that point, but go on to the generation of fibrous tissue, which replaces the old retinal structures, and eventually, by its contraction, throttles the vessels which produced it. Whilst the process of revascularisation seems to proceed at a very constant rate in all the cases recorded, the process of organisation, which is to be regarded as antagonistic, seems to vary greatly, both in rapidity and in amount. These variations must depend upon the very complex conditions of the individual cases. Hence in one case we get venous hyperæmia and hæmorrhages; in the other these may be reduced to a minimum, though they are apparently never absent. In the latter event a moderate hyperæmia passes gradually and almost unnoticeably into anæmia, with narrow vessels which are soon obliterated. The ultimate effect is the same; the

retina is transformed into fibrous tissue, in which pigmented areas mark the sites of former hæmorrhages.

YVERT.—Traité des Blessures du Globe de l'Œil, Paris, 1880. BERLIN.—K. M. f. A., ix, 1871. LEBER.—In G.-S., v, 1876. KRENCHEL.—A. f. O., xx, 1874. WAGENMANN.—A. f. O., xxxvi, 4, 1890. v. GRAEFE.—A. f. O., v, 1, 1859. H. PAGENSTECHER.—A. f. O., xv, 1, 1869. KNAPP.—A. f. A., iv, 1874. JUST.—K. M. f. A., xi, 1873. HIRSCHBERG.—A. f. A., viii, 1879. ASCHMANN.—Dissertation, Zürich, 1884. KARAFIATH.—Nagel's Jahresbericht, 1884. v. HIPPEL.—Berliner klin. Woch., 1886. SCHLIEPHAKE.—Dissertation, Giessen, 1888. LIEBRECHT.—K. M. f. A., xxix, 1891. BLESSIG.—Nagel's Jahresbericht, 1898. MENDEL.—Berliner klin. Woch., 1899. NEUBURGER.—Münchener med. Woch., 1901. BIRCH-HIRSCHFELD.—K. M. f. A., xl, 1902. *PARSONS.—R. L. O. H. Rep., xv, 4, 1903.

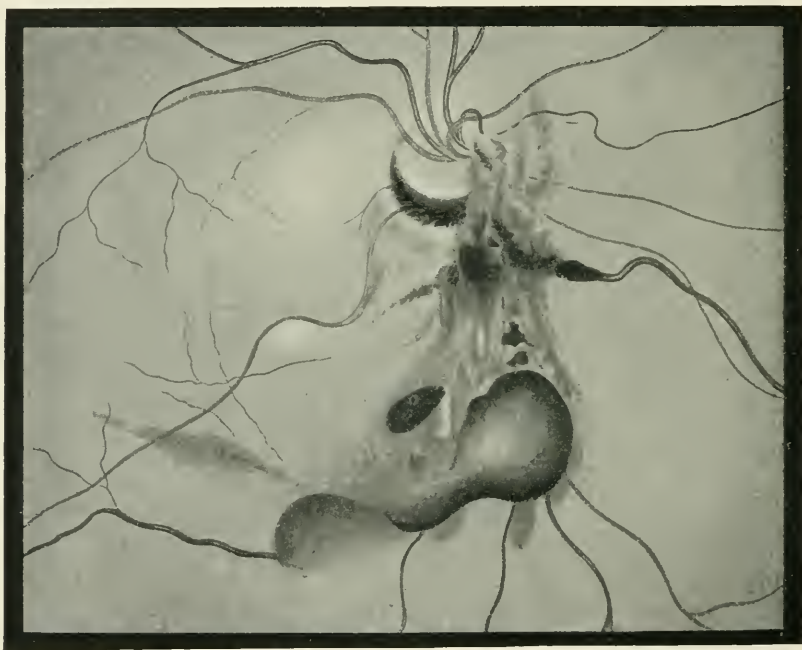Without actual severance of the optic nerve in the orbit it may be

FIG. 799.—RUPTURE OF LAMINA CRIBROSA.
Lang, T. O. S., xxi. From a boy, æt. 14, who was struck with a clothes' prop; one day after injury.

seriously injured in various ways. Shot (v. Graefe, Vossius), copper caps (Rava), stone (Hillemanns), iron (Keyser), etc., may become embedded in it. It may be contused by blunt foreign bodies, projectiles, etc. The nerve has been torn inside the sheath immediately behind the eye by the point of an umbrella (His), cow's horn (Pagenstecher), nail (Zirm), etc. The nerve has been torn out of the eye with a portion of the retina (Aschmann, Baer, Nürnberger, Gehl), less frequently out of the optic foramen (Bower, Post). The former injury may be incomplete, as in the cases of Niederhauser and Mannhardt; in the latter a false aneurism was formed from rupture

of a branch of the central artery. The nerve may be lacerated by fragments of bone, shot (Vossius, Schweigger, Leber, Keyser, and others), etc.

v. GRAEFE, HIS.—In G.-S., v, p. 916, 1876. RAVA.—Ann. di Ott., x, 1881. HILLEMANNS. —A. f. A., xxxii, 1896. KEYSER.—Ann. di Ott., viii, 1879. PAGENSTECHER.—A. f. A., viii, 1879. ZIRM.—C. f. A., xxi, 1897. ASCHMANN.—Dissertation, Zürich, 1884. BAER.—In Fischer, Handb. der Kriegschir., 1888. NÜRNBERGER.—Dissertation, Kiel, 1894. GEHL.— Dissertation, Kiel, 1888. BOWER.—Brit. Med. Jl., 1888. POST.—Amer. Jl. of O., iv, 1887. NIEDERHAUSER.—Dissertation, Zürich, 1875. MANNHARDT.—K. M. f. A., xiii, 1875. VOSSIUS. —K. M. f. A., xxi, 1883. SCHWEIGGER.—K. M. f. A., xii, 1874. SNELL AND GARRARD.— Brit. Med. Jl., 1888. SNELL.—T. O. S., xvii, 1897. GOTTBERG.—A. f. A., xxx, 1895.

Injury of the optic nerve in the scleral canal is rare and usually

FIG. 800.—RUPTURE OF LAMINA CRIBROSA.
From the same eye, about a month later.

results from a penetrating wound of the eye—*e. g.* Mauthner, wound with an arrow, Samelsohn, with a shoemaker's awl, etc. Penetrating foreign bodies may lodge in the papilla (Krüger, Hoffmann, Webster, Galezowski, Adler, Oeller, and others). Lang records a case of rupture of the lamina cribrosa and optic nerve fibres from a blow by a clothes' prop in a boy, æt. 14.

MAUTHNER.—Vörtrage a. d. Gesamtgeb. d. Augenheilk., Wiesbaden, 1878. SAMELSOHN. —B. d. o. G., 1877. KRÜGER, HOFFMANN.—B. d. o. G., 1887. WEBSTER.—Nagel's Jahresbericht, 1886. ADLER.—Prager med. Woch., 1895. OELLER.—Ophth. Atlas, pl. xiv. LANG.—T. O. S., xxi, 1901. V. MICHEL.—Z. f. A., vi, 1901. SALZMANN.—Z. f. A., ix, 1903. GAGARIN.—K. M. f. A., xlii, 1904. GENTH.—A. f. A., xlix, 1904. HESSE.—Z. f. A., xvii, 1907.

VII. Injuries of the Orbit.

No attempt will be made to treat injuries of the orbit exhaustively here. The subject is discussed by Praun and others. The chief
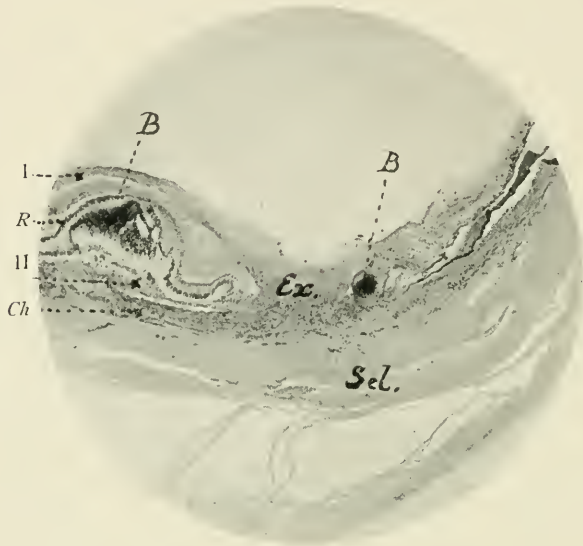

FIG. 801.—Gunshot Injury.   × 17.

Nettleship, T. O. S., xxiv. Indirect gunshot injury, showing rupture of choroid (*Ch.*) and retina (*R.*), with exudation (*Ex.*) passing between *Ch.* and *R.* (at II), and over inner surface of *R.* (at I). *Scl.* Sclerotic. *B.* Blood.

records of foreign body in the orbit, mostly remarkable for their size, nature, or duration of retention are given in the bibliography. Cases
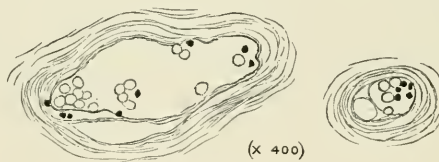

(× 400)

FIG. 802.—Gunshot Injury.   × 400.

From the same specimen. Showing minute black particles, presumably carbon, within small choroidal vessels.

of foreign body followed by tetanus are reported by Hulke, Rockliffe, Fromaget, Marx. It is interesting to note that blindness may follow injury by a bullet which does not touch the eyeball or optic nerve, probably from concussion (Cohn, Nettleship).

Zander and Geissler.—Ueber die Verletzungen des Auges, Leipzig, 1864. Berlin.— In G.-S., vi, 1880. Falch.—Dissertation, Greifswald, 1879. Lawson.—Lancet, 1877. Bower.—Brit. Med. Jl., 1879. Nicolini.—Ann. di Ott., ix, 1880. Cast.—Brit. Med. Jl., 1880. Kruger.—B. d. o. G., 1881. Noyes.—Amer. Med. Soc., 1882. v. Hippel.—Berliner

klin. Woch., 1886. COLLETTE AND ANSIEUX.—Ann. d'Oc., xxiii, 1850. BAUDRY.—A. d'O., vi, 1886. RING.—New York Med. Jl., 1887. LOTZ.—K. M. f. A., xxviii, 1890. NORRIS.—T. Am. O. S., 1890. JOHNSON.—Am. Jl. of O., xi, 1894. HÖHNE.—K. M. f. A., xxxiv, 1896. ZENKER.—K. M. f. A., xxxvi, 1898. *PRAUN.—Die Verletzungen des Auges, Wiesbaden, 1899. HULKE.—Brit. Med. Jl., 1887. ROCKLIFFE.—Brit. Med. Jl., 1890. MARX.—Dissertation, Berlin, 1893. FROMAGET.—A. d'O., xiv, 1894. COHN.—Schussverletzungen des Auges, Erlangen, 1872. NETTLESHIP.—T. O. S., xxi, 1901; xxiv, 1904. SCHOLZ.—Dissertation, Greifswald, 1905. *NICOLAI.—A. f. A., xliv, 1902. MAYWEG, JUN.—K. M. f. A., xlv, 1907. ULBRICH.—A. f. A., lviii, 1907.

Injuries of the soft parts by blunt objects often lead to extravasation of blood and proptosis (Berlin, Morian, Michards, Pagenstecher, Querenghi, Snell, Priestley Smith, Friedwald, and others). Myopia of 6 D produced by lateral compression of the eye is recorded by Ulrich. Wounds of the soft parts near the orbital margin are common, with œdema (Priestley Smith), inoculation of syphilis (Morel-Lavallée), tetanus (Fromaget, Wahl), enophthalmos (q. v.), etc. Much attention has been directed to so-called "supra-orbital amaurosis and amblyopia," said to be due to reflex irritation of the supra-orbital nerve (Platner, Beer, Middlemore, and Wallace).
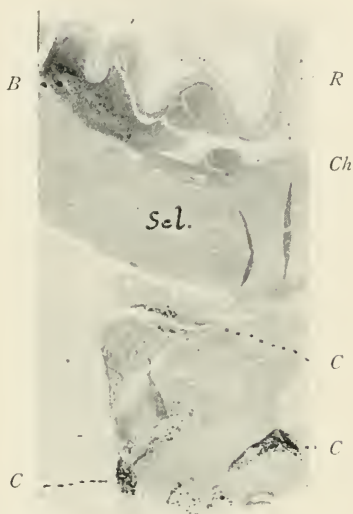

FIG. 803.—GUNSHOT INJURY. × 10.

From the same specimen. Showing particles of carbon (C.) behind sclera (Scl.); smaller particles are present in the subretinal blood clot (B.). Ch. Choroid. R. Retina. From bleached section.
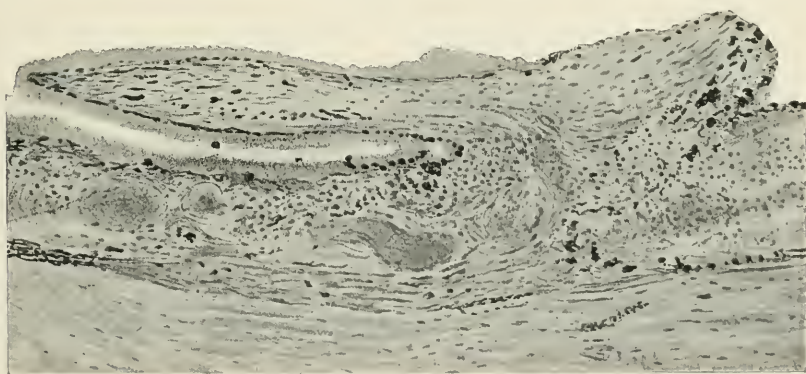

FIG. 804.—GUNSHOT INJURY. × 100.

From the same specimen. Showing fibrous tissue between pigment epithelium and external limiting membrane of retina.

Many of these cases are due to commotio retinæ, others to cerebral injury, retrobulbar neuritis, etc. Some must be attributed to neurosis

(*cf.* Leber) : they are analogous to the cases of amblyopia associated with carious teeth, phlyctenular ophthalmia (v. Graefe), etc. The subject has been investigated experimentally with no very conclusive results by Vicq d'Azyr, Romberg, Luna, Fernandez. Many cases of traumatic dislocation of the lacrymal gland have been reported (v. Graefe, Rampoldi, Haltenhoff, Bistis, Ahlström, Hilbert, Mittendorf, Villard, Coppez, Santucci, Purtscher, Jackson, Collomb and Doret).

Contusion and fracture of the orbital margin is common (Middeldorpf, Biermeyer, Mackenzie, Mooren, Gayet, Brandenburg). Fracture of the roof of the orbit, which is very thin, is a serious injury likely to lead to death from injury to the brain (Bergmann, Anders, Norton, Jones, Bock, Zinsmeister, Messerer, and others). Fracture of the inner wall is likely to be accompanied by emphysema (Baudry, Berlin, de Wecker, Marcus, and others). Fractures during spontaneous or assisted parturition are recorded by v. Hofmann, Schröder, Bloch, Danyan (breech presentation) Mackenzie, Bouchut, Lomer, and others (*see* Wolff). Shot injuries of the orbit are common (*see* Praun).

BERLIN.—In G.-S., vi, 1880. MORIAN.—Z. f. Chir., xviii. PAGENSTECHER.—A. f. A., xii, 1883. QUERENGHI.—Ann. di Ott., xix, 1890. ULRICH.—K. M. f. A., xx, 1882. PRIESTLEY SMITH.—R. L. O. H. Rep., xii, 1888. MOREL-LAVALLÉE.—Ann. de Dermat., 1886. FROMAGET. — A. d'O., xiv, 1894. WAHL. — Petersburger med. Woch., 1882. PLATNER.—De Vulneribus superciliis, Lipsiæ, 1741. BEER.—Lehre von d. Augenkrankheiten, i. LEBER.—In G.-S., v, 1877; A. f. O., xxvi, 2, 1880. v. GRAEFE.—A. f. O., xii, 2, 1866. RAMPOLDI.—Ann. di Ott., xiii, 1884. HALTENHOFF.—Ann. d'Oc.. cxiii, 1895. BISTIS.— Ann. d'Oc., cxiv, 1895. AHLSTRÖM.—C. f. A., xxii, 1898. HILBERT.—K. M. f. A., xxxviii, 1900. MITTENDORF.—T. Am. O. S., xxxvii, 1901. VILLARD.—Rev. gén. d'O., 1903. COPPEZ.—A. d'O., xxiii, 1903. SANTUCCI.—Ann. di Ott., xxxii, 1903; C. f. A., xxviii, 1904. PURTSCHER.—C. f. A., xxvii, 1903. JACKSON.—Ophth. Rec., 1904. SCRINI.—A. d'O., xxv, 1905. *COLLOMB AND DORET.—Ann. d'Oc., cxxxvi, 1906. MIDDELDORPF.—Breslauer ärztl. Z., 1886. BRANDENBURG.—A. f. A., xxxi, 1895. BERGMANN.—Die Lehre von den Kopfverletzungen, Stuttgart, 1880. NORTON.—A. of O., xiii, 1884. MESSERER.—C. f. Chir., 1884. MARCUS.—Deutsche Z. f. Chir., xxiii. v. HOFMANN.—Lehrb. d. gerichtl. Med. 1891. SCHRÖDER.—Lehrb. d. Geburtshilfe, 1884. BLOCH.—C. f. A., xv, 1891. DANYAN.—Schmidt's Jahrb., 1844. LOMER.—Z. f. Geburtshilfe, x, 1884. DITTRICH.—Wiener klin. Woch., 1892. ZACKE.—Dissertation, Berlin, 1889. TRUC.—Ann. d'Oc., cxix, 1898. FRITSCH.—Klink. d. geburtsh. Operationen, Halle, 1894. CRAMER.—C. f. Gynäk., 1899. *WOLFF.—Hirschberg's Festschrift, Leipzig, 1905. *PRAUN.—Die Verletzungen des Auges, Wiesbaden, 1899.

CHAPTER XXIII

EXOPHTHALMOS AND ENOPHTHALMOS

The causes leading to forward or backward displacement of the globe in the orbit are so various that these conditions are most conveniently discussed in a separate chapter. The best classification is that recently adopted by Birch-Hirschfeld:

1. Exophthalmos
 - A. Protrusio bulbi
   - (a) Encroachment on the orbital cavity
     - (α) Deformity of the orbital walls.
     - (β) Increase in the orbital contents.
   - (b) Diminished retraction: paralysis of VII and III
 - B. Protractio bulbi
   - Increased protraction (obliques): stimulation of sympathetic
   - Exophthalmos after tenotomy of recti. Paralytic exophthalmos.

2. Enophthalmos
 - A. Retrusio bulbi
   - (a) Pressure from in front (e.g. tumours in the anterior part of the orbit).
   - (b) Diminished protraction, paralysis of sympathetic.
     - Tenotomy of obliques.
 - B. Retractio bulbi
   - (a) Increased retraction.
   - (b) Relative or absolute increase of the orbital cavity (e.g. fracture of walls, absorption of orbital fat, cicatricial retraction).

EXOPHTHALMOS.

EXOPHTHALMOS FROM DEFORMATION OF THE ORBITAL WALLS.—Deformation of the orbital walls may be congenital or acquired; in the latter case it may be infantile or developed in later life. It is best to consider the congenital and acquired infantile varieties together, since both depend largely upon the peculiarities of development of the orbit and of its anatomy in early life.

A coronal section through the orbit of the adult shows that it is approximately circular. In the new-born child it is egg-shaped, with the long axis directed down and in, and the pointed pole down and in (Merkel and Kallius). The vertical diameter increases rapidly, and at five years of age it is only 2—3 mm. less than in the adult. Later there is some increase in breadth (Schneller). The changes which occur are chiefly associated with the development of the nasal system of bones and their sinuses. These sinuses are undeveloped or only minimally

represented at birth, so that the orbital plate of the frontal bone, which slopes down and in, forms almost the entire upper and inner boundary of the orbit. At a later stage the development of the nasal cavity and of the ethmoidal and sphenoidal sinuses raises the inner end of the orbital plate, which thus becomes horizontal, and extends the minimal inner wall of the orbit, which thus becomes an important boundary. In dealing, therefore, with congenital and infantile deformation of the orbit, the nasal sinuses, which play so large a part in later life, are almost negligible, and the cause is to be found in factors which lead to precocious or delayed union of the interosseal sutures. It will be found that congenital deformity usually arises from the former, infantile from the latter cause.

BIRCH-HIRSCHFELD.—In G.-S., ix, 1907. MERKEL AND KALLIUS.—In G.-S., i, 1901. SCHNELLER.—In Merkel and Kallius.

Congenital deformation : scaphocephaly and oxycephaly.— Scaphocephaly (Turmschädel or tower-skull) and oxycephaly (Spitzkopf)

FIG. 805.—OXYCEPHALY.
Donaldson, T. O. S., xxiii.

are conditions dependent upon precocious union of the coronal suture. Oxycephaly was attributed to this cause by Virchow, scaphocephaly by Enslin. In the latter condition the compensatory elevation of the cranial dome is insufficient and lateral extension takes place below. Hence the very marked protrusion of the temporal region, often with the development of definite bosses and bowing out of the zygomata. The most important effect on the orbit is the change in direction of the great wing of the sphenoid (Weiss and Brugger), which, from normally entering into the median wall, becomes almost coronal in plane and forms a posterior boundary. The orbital space is thus encroached upon, though to a very different degree in various cases : hence, the

variation in the amount of exophthalmos (v. Michel, Schüller, Hirschberg, Vossius, Manz, Weiss and Brugger, Groenouw, Vortisch, Friedenwald, Kraus, Taylor, Uhthoff, Patry, Paton, Stephenson, and others). The coronal section is vertically oval (Weiss and Brugger), though this is only so posteriorly (Enslin). The exophthalmos may be extreme (Uhthoff, Stephenson), even to the extent of causing keratitis e lagophthalmo and loss of the eye thereby (Uhthoff). In Stephenson's case there was good evidence of heredity—narrow orbits and prominent

Fig. 806.—Oxycephaly.
Power, T. O. S., xiv.

eyes in the mother, exophthalmos and death from fits in a younger sister : heredity is also noted in the cases of Carpenter and Velhagen. Power and Swanzy report anatomical examinations. In Power's case the orbital plate of the frontal bone was almost vertical; in Swanzy's it made an angle of 45° with the horizontal floor.

Optic neuritis (v. Graefe, Enslin, Manz, Kampherstein) and optic atrophy, postneuritic or primary, are recorded in several of the cases, and are to be attributed to the deformity of the optic foramen and intra-cranial causes ; they are not immediately associated with the exophthalmos.

VIRCHOW.—Die Entwickelung des Schädelgrundes, Berlin, 1857. v. GRAEFE.—A. f. O., xii, 1866. v. MICHEL.—A. f. Heilk., 1873; Gerhardt's Handb. d. Kinderkrankh., 1889. SCHÜLLER.—C. f. A., v, 1881. HIRSCHBERG.—C. f. A., vii, 1883. VOSSIUS.—K. M. f. A., xxii, 1884. MANZ.—B. d. o. G., 1887. FRIEDENWALD.—Amer. Jl. of Med. Sc., 1893; A. of O., xxx, 1901. POWER.—T. O. S., xiv, 1894. WEISS AND BRUGGER.—A. f. A., xxviii, 1894. NETTLESHIP.—T. O. S., vii, 1887; xxv, 1905. SWANZY.—T. O. S., xviii, 1898. GROENOUW.—In G.-S., xi, 1, 1901. CARPENTER.—Soc. for Children's Diseases, i, 1901. VORTISCH.—Dissertation, Tübingen, 1901. KRAUS.—Dissertation, Giessen, 1902. DONALDSON.—T. O. S., xxiii, 1903. *ENSLIN.—A. f. O., lviii, 1, 1904. TUCKER.—Lancet, 1904.

FIG. 807.—OXYCEPHALY.
From the same specimen.   Antero-posterior section through the right eye.

MORAX AND PATRY.—Ann. d'Oc., cxxxii, 1904. TAYLOR.—Soc. for Children's Diseases, iv, 1904. VELHAGEN.—Münchener med. Woch., 1904. AMBIALET.—Ann. d'Oc., cxxxiv, 1905. OBERWARTH.—A. f. Kinderheilk., xlii, 1905. UHTHOFF.—K. M. f. A., xliii, 1905. PATRY.—Thèse de Paris, 1905; Ann. d'Oc., cxxxiii, 1905; cxxxv, 1906. PATON.—T. O. S., xxv, 1905; xxvii, 1907. STEPHENSON.—Soc. for Children's Diseases, v, 1905. KAMPHERSTEIN.—K. M. f. A., xliii, 1905. COHEN.—K. M. f. A., xliv, 1906. COATS.—T. O. S., xxvii, 1907. FORD.—Ophthalmoscope, 1907. KRAUSS.—Z. f. A., xvii, 1907.

Infantile deformation.—Exophthalmos has been observed in cases of hydrocephalus and rickets, or a combination of both.   It is rare in uncomplicated hydrocephalus, but Uhthoff has published cases and it is mentioned by Heineke.   Probably most surgeons attached to a children's hospital have seen it occasionally.   The eye is displaced forwards and downwards, so that it may be covered by the lower lid. The rarity is doubtless due to the fact that the interosseal sutures are unclosed and the cranial cavity expands under the increased internal pressure.   It is interesting to note in this connection that in one of Uhthoff's cases there had been otorrhœa which had led to thickening of the anterior part of the skull : the exophthalmos was extreme and

the right eye was lost from keratitis e lagophthalmo.   Optic neuritis appears to be commoner in these cases than in hydrocephalus without exophthalmos.

Exophthalmos is seldom present in rickets, but the cases of Schapringer, Groenouw, and Cohen seem unexceptionable.   In Schapringer's case the proptosis was unilateral ; in one of Groenouw's the skull was very asymmetrical, and proptosis was greater on the left side.   Groenouw's second case was a combination of hydrocephalus and rachitis.   Cohen's case was interesting from the resemblance to tower skull, though the coronal suture was not ossified.   Each eye lay

FIG. 808.—EXOPHTHALMOS DUE TO CAVERNOUS ANGIOMA OF ORBIT.
W. T. Holmes Spicer, T. O. S., xxiii.

like a ball on a plate, and separation of the lids caused luxation of the eye and lacrymal gland.   The cause of exophthalmos in rickets may be due to the not very uncommon association with hydrocephalus, or to periosteal thickening such as occurs elsewhere in the disease.   Some cases may be due to subperiosteal hæmorrhage (cf. Holmes Spicer).

Other peculiar deformities of the skull associated with exophthalmos are discussed by Ambialet, the chief sign being depression of the frontal region.

UHTHOFF.—K. M. f. A., xliii, 1905.   HEINEKE.— In Billroth and Luecke, Deutsche Chirurgie, 1882.   SCHAPRINGER.—New York Med. Woch., xiii, 1900.   GROENOUW.—In G.-S., xi, 1, 1902.   COHEN.—K. M. f. A., xliv, 1906.   AMBIALET.—Ann. d'Oc., cxxxiv, 1905.

Later acquired deformation of the orbit is due to inflammatory or neoplastic swelling in the bones or sinuses. Only the acute and chronic inflammatory affections of the sinuses will be considered here. Acute and chronic inflammation in any of the accessory sinuses of the nose may produce ectasia leading to exophthalmos.

Ectasia of the *frontal sinus* is the commonest and most easily recognised, though it not infrequently gives rise to difficulty in diagnosis. It is important to remember that this sinus does not attain appreciable size until the second year of life, and that its size varies within wide limits in different skulls and on the two sides of the same skull. The thinnest part of its wall forms the boundary of the

Fig. 809.—CAVERNOUS ANGIOMA OF ORBIT.
W. T. Holmes Spicer, T. O. S., xxvi. Cavernous angioma of orbit causing extreme proptosis.

inner and upper part of the orbit, corresponding approximately with the position of the equator of the globe. Ectasia is accompanied by displacement of the globe forwards, downwards and outwards. The movements of the globe are affected not only by the displacement and diminution of space, but also by the displacement of the trochlea of the superior oblique. Acute empyema of the sinus causes the most rapid ectasia, and is most likely to discharge into the orbit. Chronic mucocele of the sinus may lead to the most extreme exophthalmos (Hallauer, Langenbeck-Barkhausen). Optic neuritis or atrophy have been described, usually in association with other complications or involvement of other sinuses (Martin, Trombetta, Cross, Desbrières, Elschnig, König, Leber, and others). Keratitis e lagophthalmo does not occur, owing to simultaneous displacement of the lids, whereas it

is common in otherwise similar displacement from tumours. Empyema may burst into the orbit and set up cellulitis, or into the cranial cavity and thus cause death (Cross, Desbrières, Kuhnt, Hoppe, Knapp, Leber).

LANGENBECK-BARKHAUSEN.—Neue Bibliothek f. d. Chir. u. Ophth., ii, 1819. KNAPP.—A. f. A., ix, 1880. LEBER.—A. f. O., xxvi, 3, 1880. HIGGENS.—Guy's Hosp. Rep., 1881. BULL, KIPP.—T. Am. O. S., 1885. REEVE.—Canadian Practitioner, 1887. ELSCHNIG.—Wiener med. Woch., 1888. WALKER.—R. L. O. H. Rep., xii, 1889. WILLIAMS.—Lancet, 1890. HULKE.—Lancet, 1891. CROSS.—Ophth. Rev., xi, 1892. HOPPE.—K. M. f. A., xxxi, 1893. COLLIER.—Lancet, 1894. GRIFFITH.—T. O. S., xiv, 1894 ; xx, 1900. KUHNT.—Ueber die entzündl. Erkrankungen d. Stirnhölen, Wiesbaden, 1895. DESBRIÈRES.—Ann. d'Oc., cxx, 1898. HALLAUER.—Z. f. A., ii, 1899. JESSOP.—T. O. S., xxi, 1901. EVERSBUSCH.—In G.-S., ix, 1903. DENNIS, WHITEHEAD.—A. of O., xxxiii, 1904. MILLIGAN.—Brit. Med. Jl., 1905. BOSSALINO.—Ann. di Ott., xxxiv, 1905. *BIRCH-HIRSCHFELD.—In G.-S., ix, 1907 (Bibliography).

Ectasia of the *ethmoidal sinuses* probably occurs more often than is diagnosed. The posterior cells not infrequently bound the optic foramen (Fuchs, Stanculéanu, Onodi), so that it is not improbable that some obscure cases of unilateral optic neuritis or atrophy may be due to this cause (*cf.* Jessop) : the inflammation may indeed be limited to these cells (Birch-Hirschfeld). The disease may be quite painless (Ahlström, Vieusse, and others) and the nose may be normal (Ahlström and others). Definite ectasia usually manifests itself in swelling near the internal palpebral ligament, which may be as hard as bone (Ahlström, Sattler), or give fluctuation (de Schweinitz). Chronic mucocele may cause enormous swelling, even to the size of a fist, with 200 grms. of fluid contents (Steiner). Periodic exophthalmos may occur (Eversbusch), and exophthalmos may be due to Tenonitis (Rohmer). Empyema may lead to orbital cellulitis (Juffinger, Vieusse, Hajek, Posey).

GRUENING.—New York Eye and Ear Hosp. Rep., i, 1895. ROHMER.—Soc. franç. d'O., 1895. POOLEY.—Am. Jl. of O., 1897. STEINER.—C. f. A., xxi, 1897. AHLSTRÖM.—K. M. f. A., xxxvi, 1898. JUFFINGER.—Wiener klin. Woch., 1898. VIEUSSE.—Rec. d'O., 1898. *HAJEK.—Path. u. Ther. d. entzündl. Erkrankungen d. Nebenhohlen der Nase, Leipzig, 1899. SATTLER, DE SCHWEINITZ.—Ophth. Rec., 1899. WEINHOLD.—K. M. f. A., xxxviii, 1900. POSEY.—Ophth. Rec., 1902. STANCULÉANU.—A. d'O., xxii, 1902. EVERSBUSCH.—In G.-S., ix, 1903. ONODI.—Z. f. A., xii, 1903. JESSOP.—T. O. S., xxiii, 1903. FUCHS.—Lehrbuch, 1905. PAUNZ.—A. f. A., lii, 1905. CIRINCIONE.—K. M. f. A., xliv, Beilageheft, 1906. *BIRCH-HIRSCHFELD.—In G.-S.,·ix, 1907 (Bibliography).

Ectasia of the *sphenoidal sinuses* is still more difficult to diagnose, but even more important, owing to their immediate relationship to the optic foramen. Moreover the walls may be extremely thin (Berger and Tyrmann). The sinuses show great individual variations, and both may be involved (Fuchs). Exophthalmos is caused by inflammation of the orbital tissues, not by the ectasia, unless other sinuses are also attacked (*e.g.* Hoffmann). Exophthalmos with retrobulbar neuritis is reported by Greene and Würdemann. Tumours in this situation may of course cause mechanical exophthalmos (Chisholm, Dombrowski, Morax, Vogel). Meningitis or cerebral abscess may occur. It is noteworthy that Fränkel found affection of the sphenoidal sinuses in 63 cases out of 146 autopsies, and Harke in a still greater percentage.

CHISHOLM.—A. of O., xi, 1882. BERGER AND TYRMANN.—Wiener med. Bl., 1886. SCHAEFFER.—Deutsche med. Woch., 1892. DOMBROWSKI.—Am. Jl. of O., xii, 1895.

HOLMES.—A. of O., xxv, 1896. MORAX.—Soc. franç. d'O., 1896. GREENE.—Ophth. Rec., 1897. AVELLIS.—Deutsche med. Woch., 1903. WÜRDEMANN.—Ophth. Rec., 1905. JESSOP. —T. O. S., xxiii, 1903 ; xxiv, 1904. *BIRCH-HIRSCHFELD.—In G.-S., ix, 1907 (Bibliography).

Ectasia of *several of the accessory sinuses of the nose* is frequent owing to their communication with each other. Involvement of the frontal and the ethmoidal (Bryan, Knapp, Peters, Harlan, Johnston and others) and of the ethmoidal and sphenoidal (Berger and Tyrmann, Miller, and others) is most common, or all three may be affected (Harlan Peters).

BERGER AND TYRMANN.—Die Krankheiten der Keilbeinhöhle, etc., Wiesbaden, 1886. NIEDEN.—A. f. A., xvi, 1886. DE LAPERSONNE.—A. d'O., xviii, 1897 ; Ann. d'Oc., cxxvii, 1902. BRYAN.—Ophth. Rev., xviii, 1899. GRADLE.—Ophth. Rec., 1899. KNAPP.—A. f. A.,, xxxix, 1899 ; A. of O., xxxii, 1903. PETERS.—Z. f. A., ii, 1899. HARLAN, RISLEY.—T. Am. O. S., 1900. MILLER.—Brit. Med. Jl., 1900. SATTLER.—Ophth. Rec., 1900. MENDEL.— C. f. A., xxv, 1901. AXENFELD.—Deutsche med. Woch., 1902. JESSOP.—T. O. S., xxiii, 1903. OLIVER AND WOOD.—Am. Jl. of Med. Sc., 1902. ONODI.—Z. f. A., xii, 1904. JOHNSTON.— Ophth. Rec., 1905. PAUNZ.—A. f. A., lii, 1905. HOFFMANN.—Z. f. A., xvi, Ergänzungsheft, 1906. CRAMER.—K. M. f. A., xliv, Beilageheft, 1906. *BIRCH-HIRSCHFELD.—In G.-S., ix, 1907 (Bibliography).

Ectasia of the *maxillary antrum* by inflammatory processes scarcely ever produces exophthalmos. Orbital cellulitis not infrequently arises from empyema of the antrum of Highmore, causing proptosis (Burnett, Merz, Panas, Bauby, Rollet, Deschamps, Roure, Guttmann, Ogchu, and others). Tumours are more likely to cause ectasia of the upper wall or break through into the orbit (Benson, Harlan, Puccioni).

BENSON.—Brit. Med. Jl., 1882. BURNETT.—A. of O., xiv, 1885. MERZ.—K. M. f. A., xxxiii, 1895. PANAS.—A. d'O., xv, 1895. BAUBY.—A. d'O., xvii, 1897. ROLLET.—Ann. d'Oc., cxviii, 1897. DESCHAMPS, ROURE.—A. d'Oc., cxx, 1898. HARLAN.—Ophth. Rec., 1898. GUTTMANN.—C. f. A., xxiii, 1899. PUCCIONI.—A. ital. di Otol., ix, 1899. GRADAILLE. —K. M. f. A., xl, 1902. OLIVER AND WOOD.—Am. Jl. of Med. Sc., 1902. OGCHU.— Annals of Ophth., 1903. FISH.—A. f. A., lii, 1905. GALEZOWSKI.—Rec. d'O., 1905. HOFFMANN.—Z. f. A., xvi, Ergänzungsheft, 1906. GREEN.—Ophth. Rec., 1906. *BIRCH-HIRSCHFELD.—In G.-S., ix, 1907.

EXOPHTHALMOS FROM INCREASE IN THE ORBITAL CONTENTS may be due to foreign bodies, hæmorrhage, orbital cellulitis, tumours, etc. These subjects are considered elsewhere. Exophthalmos in *acromegaly* is most likely to be attributable to this cause. It is found noted by Hertel in seventeen cases, and may be extreme (Maisonneuve, Mastri, Trachtenberg). It may be associated only with feverish attacks (Motais), or may spontaneously disappear (Orsi). These cases suggest that it may be due to pressure on the cavernous sinus and impediment to return of blood from the orbit (Orsi). Hertel considers that it is due to increase in orbital fat, but there is no anatomical support for this conjecture. In some cases it is probable that it is caused by hypertrophy of the bones. Acromegaly may also cause apparent enophthalmos, probably due to hypertrophy of bone manifesting itself in the anterior part of the orbit (Gauthier, Maissonneuve, Gordon Brown and others).

MOTAIS.—Ann. d'Oc., xcv, 1886. APPLEYARD.—Lancet, 1892. GORDON BROWN.— Brit. Med. Jl., 1892. HERTEL.—A. f. O., xli, 1, 1895 (Bibliography). MARCUS GUNN.— T. O. S., xvi, 1896. HUTCHINSON.—New York Med. Jl., 1900. CROSS.—Brain, 1902. TRACHTENBERG.—Z. f. klin. Med., xlii, 1902. PORTER.—Ophth. Rec., 1906. *BIRCH-HIRSCHFELD.—In G.-S., ix, 1907 (Bibliography).

INTERMITTENT EXOPHTHALMOS.—This designation has been applied to various conditions of temporary exophthalmos, such as those caused by orbital hæmorrhage, angioma, etc. It should be reserved for cases in which proptosis is caused by stooping, compression of the jugular vein, forced expiration, etc., and disappears or is replaced by enophthalmos when such cause ceases to act. With this limitation the condition is extremely rare. Only one case has been seen at the Leipzig clinic in sixteen years—i. e. amongst about 150,000 patients, and Birch-Hirschfeld has found only fifty cases in the literature. The disease may manifest itself at any age. The cases of Magnus, Lang and Thompson, Priestley Smith, and Hirschmann appear to date from birth : during the first decade, Schwarzchild, Sergent, Radswitzky, Meyer, Posey ; second decade, Gessner, Treacher Collins, Becker, Richter, Lang, Moyart and van Duyse, Mulder ; third decade, Dunkel, Elschnig, van Duyse and Bribosia, Terson, Lesshaft, Ostrowitzky, Minor ; fourth decade, Kohl ; fifth decade, Grüning, Schmidt-Rimpler. Sattler's opinion that it was essentially a disease of early life is therefore not confirmed. It occurs more commonly in men, in the proportion 35 to 15 (Birch-Hirschfeld).   It is invariably unilateral.   The exophthalmos is often stated to have come on suddenly, during exertion (Grüning, Mackenzie, Elschnig, Meyer) or trauma (Schwarzchild, Treacher Collins, van Duyse and Bribosia, Priestley Smith, Voigt). Too much importance has probably been attached to the latter factor, and it is not improbable that a gradual onset is commoner than the records would appear to show. The most accurately and exhaustively investigated case is that of Minor and Birch-Hirschfeld.

The amount of proptosis varies in different records from 4—5 mm. to 25 mm., as in Sattler's case ; most of these results are mere computations, not exact measurements. The latter have been made only in Minor's case by a photographic method—on compression of the jugular vein 11·44 mm., on forced expiration 5·72 mm., on bending the head back laterally so that the jugular vein was compressed by the active sterno-cleido-mastoid 14·3 mm. ; on bending the head forwards the proptosis increases very rapidly, the maximum of nearly 17 mm. being reached in forty-five seconds. The proptosis may be directly forwards, forwards and downwards (Magnus, Radswitzky, Kooyker and Mulder, Minor), or forwards and upwards (Richter). The deviation from the axial direction depends probably on the relative overfilling of the superior or inferior ophthalmic vein. There is usually also swelling of the lids and conjunctiva, and cerebral symptoms are described (Dunkel, Treacher Collins, Trombetta, Schmidt-Rimpler, Posey, Surow, Kohl).

The retinal veins are dilated and pulsate during the proptosis, but otherwise the fundus is usually normal. Optic atrophy is described by van Santen, Mayer, Lang, Lang and Thompson, all probably due to retrobulbar hæmorrhage. Pallor of the disc is mentioned by Elschnig, Grüning, and Lesshaft. Cloudiness of vision during exophthalmos is described (Vieusse, Gessner, van Duyse and Bribosia, Lang and Thompson, Lesshaft, Posey, Surow, Kohl), but may be absent (Dunkel, Treacher Collins, Minor).   The pupils may be dilated (Becker, Terson,

van Duyse and Bribosia) ; this cannot well be due to stretching of the ciliary nerves (van Duyse and Bribosia), as it should then be a constant feature of exophthalmos.

In many cases there is slight enophthalmos when the head is upright, generally in old standing cases (Sattler, Gessner, Schwarzchild [6 mm.], Sergent, Treacher Collins, van Duyse and Bribosia [4 mm.], Mayer, Radswitzky [2—3 mm. in the upright, 5—6 mm. in the supine position], Meyer, Mulder, Hitschmann, Minor). It is probably due to absorption of orbital fat.

Varicosities in the vessels in other parts of the body are described (Magnus, angiomatous tumours of face and head; Dunkel, hæmorrhoids, angiomata of the skin : Mayer, Kooyker and Mulder, Meyer, Leitner, Hitschmann [including mouth], Kohl [of the conjunctiva]). In several the varices affected the jugular vein area (Magnus, Leitner, Hitschmann, Kohl). Facial asymmetry is recorded (Schwarzchild, Radswitzky, Kohl, Mulder, Surow, Minor). Retrobulbar hæmorrhage is a not very uncommon complication (Becker, Meyer, van Santen, Alger, Lang, Lang and Thompson). Pulsation of the eye, simulating pulsating exophthalmos (q. v.), is rare (de Vincentiis, Mulder); Grunert's and Jocq's cases were probably pulsating exophthalmos.

Many theories of the pathogenesis of intermittent exophthalmos have been advanced. Vieusse (1878) suggested communication between the arachnoid space and Tenon's capsule, but withdrew the theory later (1898). Kooyker and Mulder suggested congenital defect in the bones of the orbit. Venous tumours were conjectured by Magnus, Treacher Collins, Surow, and Voigt, especially on account of slight persistence of exophthalmos in the upright position. An inflammatory origin, advanced by Elschnig, Pick, and Jacobi, is unlikely from the absence of any signs of inflammation. Trophoneurosis of the sympathetic, suggested by Terson chiefly on the occasional presence of enophthalmos, lays too much stress on this inconstant factor, and has little in its favour. Birch-Hirschfeld advances the most likely theory, viz. partial or complete blockage of the communication of the orbital veins with the jugular vein by way of the angular and facial veins. The blockage may be anywhere between the origin of the angular vein and the entrance of the facial vein into the jugular. The absence of valves in the orbital veins supports the view that in the stooping posture the orbital blood escapes chiefly through the angular vein, whilst in the upright and supine postures it escapes into the cavernous sinus and pterygoid plexus.

Schmidt.— Ophth. Bibliothek, iii, 1805. Mackenzie. — Treatise, London, 1854. Grüning.—A. f. A., iii, 1873. Vieusse.—Rec. d'O., 1878; in Teillais, Soc. franç. d'O., 1898. Magnus.—K. M. f. A., xxii, 1884; xxvii, 1889. Sattler.—Amer. Jl. of Med. Sc., 1885. Gessner.—C. f. A., xiii, 1889. Chisholm.—A. f. A., xxii, 1891. Elschnig.—Allg. Wiener med. Zeitung, 1892. Schwarzchild.—Med. Rec., 1892. Becker.—A. f. O., xli, 1, 1895. Treacher Collins.—T. O. S., xv, 1895. van Duyse and Bribosia.—A. d'O., xv, 1895. Richter.—A. f. A., xxxi, 1896. Lang and Thompson.—T. O. S., xvii, 1897. Vossius.—B. d. o. G., 1897. Grunert.—Ophth. Klinik, 1898. Kooyker and Mulder.— Z. f. klin. Med., 1898. Lesshaft.—C. f. A., xxii, 1898. Meyer.—K. M. f. A., xxxvi, 1898. Mulder.—K. M. f. A., xxxviii, 1900. Bickerton.— T. O. S., xx, 1900. Hitschmann.— Wiener klin. Woch., 1900. Schmidt-Rimpler.—Deutsche med. Woch., 1900. Posey.— Ophth. Rec., 1902 ; 1904; 1905. Voigt.—Münchener med. Woch., 1904. Jacobi.—Disser-

tation, Königsberg, 1906. MINOR.—Dissertation, Leipzig, 1907. *BIRCH-HIRSCHFELD.—
In G.-S., ix, 1907 (Bibliography).

PULSATING EXOPHTHALMOS.—Typical pulsating exophthalmos is due to arterio-venous aneurysm involving the internal carotid artery and the cavernous sinus. There is some doubt as to whether carotid aneurysm alone can cause the condition (Rivington, Le Fort, Barnard and Rugby, Beadles). Travers (1813) first accurately described the clinical features. Sattler collected and tabulated all the cases up to 1880. Two groups of cases may be distinguished, the traumatic and the spontaneous. In the latter the onset often follows some unusual exertion, such as parturition, a fit of coughing, etc.: syphilis plays an important part in the ætiology of these cases. Trauma is usually of such a nature as to render fracture of the base of the skull likely, such as fall on the head, on the feet or buttocks from a height, etc.; less commonly it is a direct injury to the orbit by a blow, gunshot, etc. The onset is generally sudden. Of 32 spontaneous cases, 23 occurred in women, 6 in men; of 59 traumatic cases, 44 occured in men, 13 in women (Sattler): most of the spontaneous cases commenced between thirty and fifty years of age.

Jack has recently reported a case, examined anatomically by Verhoeff. He states that 260 cases have been recorded and 33 autopsies, his case being the seventh in which a rupture of the carotid into the cavernous sinus has been absolutely proved. The results of the autopsies are as follows: aneurysm of the ophthalmic artery in the orbit, 2; aneurysm of the ophthalmic artery before its entry into the orbit, 1; rupture of the carotid artery into the cavernous sinus, 7. To this number may be added three reviewed by Sattler and one reported by Brandes, in which arterio-venous aneurysm was practically proved. The difficulty of demonstrating the lesion is considerable, so that probably it has been overlooked in some cases. Aneurysm of the internal carotid was present in three cases, and in another the wall was much thinned. Tumours caused five cases, and internal hydrocephalus and orbital encephalocele one each; in one there was a communication between some branches of the ophthalmic artery and the sinus. In six no aneurysm or arterial lesion was found.

Rivington made the statement that true pulsating exophthalmos could not be caused by a circumscribed or fusiform aneurysm of the carotid, contrary to the opinion of Le Fort that aneurysm of the ophthalmic artery, aneurysm of the carotid in the cavernous sinus, and rupture of the carotid into the sinus gave identical symptoms. Barnard and Rugby reported an autopsy of a typical case in which there was a double sacculated aneurysm in the intra-cranial course of the carotid without any communication with the sinus and without dilatation of the sinus or ophthalmic vein. Attention has been principally devoted to the treatment of the cases (Stowman, Reuchlin and others).

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EXOPHTHALMIC GOITRE (*Graves's disease, Basedow's disease*).—This disease was recognised by Graves in 1835, independently by Basedow in 1840. The familiar symptom-complex of this disease includes proptosis, enlargement of the thyroid gland, tachycardia, and muscular tremors. The proptosis is almost always bilateral and may be extreme, leading to lagophthalmia and its deleterious consequences. There is a peculiar stare, with retraction of the upper eyelid (Dalrymple's sign). Normally when vision is directed downwards, the upper lid moves concordantly with it : in this disease the upper lid follows tardily or not at all (v. Graefe's sign). There is diminished frequency of winking and imperfect closure of the lids during the act (Stellwag's sign). There may be imperfect power of convergence (Möbius' sign), and often the skin of the eyelids shows pigmentation (Jellinek). Ophthalmoscopically veins and arteries may be somewhat distended, but specific signs are absent.

Women are more often affected than men (46 to 1, Buschan), but the disease is more severe in the latter (Schmidt-Rimpler). The exophthalmos may be so extreme as to cause dislocation of the globe in advance of the lids (Pain). Unilateral proptosis has been reported by Fridenberg, Sichels, Völkels, Prael, Mauthner, Mooren, Rösner, Emmert, de Giovanni, Barella, Hitscham, Mandel, Trousseau, Bistis. Greater exophthalmos on one side is commoner. v. Graefe's sign is not pathognomonic (Sharkey, Hughlings Jackson, Wilbrand and Saenger and others) and may be absent (Mannheim, Russell Reynolds, Pässler, Bruns, Flatan, Murray). It may be induced in the normal subject by instillation of cocain (Kocher, Jessop). Paralyses of extra-ocular muscles are not uncommon : monographs on this feature have been written by Ballet, Liebrecht, Buschan, Mannheim, Möbius. Corneal complications are rare and are due to lagophthalmia (Craig, Jessop, and others). Berger called attention to epiphora as an initial symptom, and Vernean and Herger have reported similar cases. Berger attributes it to reflex excitation of the sympathetic vasomotor fibres, Sattler and others to irritation of the exposed cornea and imperfect removal of tears through infrequent winking. In cases of long standing there is usually diminished secretion of tears, probably due to loss of sensibility of the cornea and conjunctiva.

Œdema of the lids may be present (Mackenzie, Stellwag). This must not be confounded with the condition of the lids in the rarer cases in which signs of coincident myxœdema are observed.

Anatomical examination affords little evidence of the cause of the

proptosis. It is probably due to venous dilatation, with perhaps some lymphatic exudation. Müller's orbital muscle is quite rudimentary in man and can have little effect. The thyroid gland shows great vascular dilatation and increase in the amount of secreting tissue. There is evidence that there is not only increased secretion but also alteration in the character of the secretion. Changes in the cervical sympathetic were early described (Virchow, Knight, and others) : they consist chiefly in pigmentation of the cells, a condition frequently found as an ordinary senile change (Hale White). Changes have also been described in the medulla oblongata (Mendel), or hæmorrhage in the fourth ventricle and neighbourhood of the vagus nucleus (F. Müller).

Many theories of exophthalmic goitre have been advanced. Koeben (1855) first attributed it to disease of the sympathetic following enlargement of the thyroid gland. Eulenberg and Guttman (1873) pointed out that it could not be simply due to pressure. Many of the symptoms are undoubtedly sympathetic in origin, such as the exophthalmos, redness of the skin and increased sweat secretion, tachycardia, etc. The absence of change in the size of the pupils is striking. Claude Bernard found that the origin of the pupillary fibres was different from that of the vasomotor fibres, and on the strength of this Geigel placed the causal lesion in the medulla and upper cervical region, attributing the tachycardia to paralysis of the cardio-inhibitory fibres of the vagus. Experiments from this point of view were made by Filehne and Bienfait, but are inconclusive. Möbius (1887) stated the theory which has received most acceptance, viz. that the disease is due to over-activity of the thyroid gland. Probably there is not only increased but also disordered secretion; indeed in some cases—those with signs of myxœdema—it is likely that the normal secretion is in abeyance and that the resulting signs are due entirely to an abnormal secretion. The theory does not explain the mechanism of production of the individual symptoms, but there can be no doubt that in this the sympathetic plays a prominent part. The theory is supported by the production of exophthalmos by over-use of thyroid extract (Béclère, Edmunds), and experiments in which cocain has been administered to animals are interesting in this relationship (Jessop, Cunningham, Edmunds).

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ENOPHTHALMOS.

TRAUMATIC ENOPHTHALMOS is a rare condition. Birch-Hirschfeld has collected and collated seventy-one cases out of the literature; only four occurred in the Leipzig eye clinic in sixteen years amongst about 150,000 patients. Males were affected in fifty-seven cases, females in eight. Most were middle aged: one in the first decade (Schapringer), ten in the second, fifteen in the third, ten in the fourth, fourteen in the fifth, three in the sixth, and one in the seventh (Maklakow). The injuries were usually severe, from falls, blows with blunt objects (cow's horn), etc. Symptoms of fracture of the skull are not infrequent, though bleeding from the nose is often referable to direct injury to the nasal bones (Ogilvy, Lederer, Roux). Fracture of the malar bone is mentioned by Gessner, Daulnoy, Luniewsky, Birch-Hirschfeld and Meltzer. Fractures of the orbital margin are common, usually below (Maklakow, Morton, Cohn, Franke, Birch-Hirschfeld and Meltzer, Chaillous); down and in (Beer, Ogilvy, Daulnoy, Causé, Birch-Hirschfeld and Meltzer), outwards (Bruner, Neulen), up and up and in (Lederer). Absence of this injury is specifically denied in some cases (Denig—fall, Manz—fall, Webster, Treacher Collins—blow with horse's head, Shoemaker, Rohmer—blow with horse's head, Bistis—fall from a horse). Bony lesion was definitely present in twenty-two cases, probable in thirteen, improbable in twenty-two, almost certainly absent in eight.

Enophthalmos may be observed at any time after the injury from six hours (Schapringer, Hansell) to one year (Treacher Collins). The degree of enophthalmos varies considerably: in thirty-one cases it was 3—5 mm.; higher grades were: Löw 6 mm., Beer and Daulnoy 7 mm., Lang and Franke 8 mm., Cohn 8—9 mm., Franke and Burnett 10 mm. The measurements were probably rough. The palpebral aperture is narrowed, the movements of the lids not affected (Nieden, Gessner, Beer, Cause), or only mechanically impeded (Darier, Grunert, Löser). Defects of movement of the globe are common (upwards twenty-two cases, outwards nineteen, downwards ten, inwards five, all movements seven, all but inwards three), but may be absent (fifteen cases).

Vision was normal in thirty-four cases, nearly or quite lost by optic atrophy in fourteen. In eight of the latter there was definite fracture of the orbit. The picture of embolism of the central artery has been seen (Zimmermann), optic neuritis (Praun, Roberts), macular changes (Shoemaker, Daulnoy, Lederer), retinal hæmorrhages (Chaillous), dilatation of retinal veins (Rohmer, Talko, Schapringer), blurring of the edges of the disc (Beer, Bruner, Webster, Neulen [with choroidal rupture]). In one of Gessner's cases there were hyaline bodies on the disc (q. v.). Central scotoma in some cases gave evidence of retrobulbar neuritis (Beer, Roux, Birch-Hirschfeld, Daulnoy).

The condition of the pupil is important on account of the sympathetic nerve theory. It was normal in twenty cases: dilated and immobile—Löw (optic atrophy), Roberts (optic neuritis), Maklakow, Daulnoy, Panas, Luniewski, Roux; in these there was generally a

cause in intra-ocular or retrobulbar changes.   Loss of reaction to light
with some retention for accommodation is noted by Franke, Flemming
(5 mm., vision normal), Treacher Collins.   Constriction of the pupil
was noted by Fuchs and Neulen ; slow reactions by Lang, Schapringer,
Rohmer, and Luniewski.   Pupillary defects are often attributable to
direct injury to the globe—Beer, iridodialysis ; Morton, detachment of
retina ; Maklakow, dislocation of lens, etc. ; Birch-Hirschfeld, irido-
plegia.   The pupil reacted promptly to cocain in the cases of Flemming
and Cause.   Accommodation was normal in several cases, paralysed
from injury to the globe in some (Lang, Beer, Micas), not recorded in
most.   Diminution of intra-ocular tension (Schapringer, Morton,
Treacher Collins, Lederer, Birch-Hirschfeld) probably depends upon
contusio bulbi.

Defects of sensibility in the regions supplied by the fifth nerve are
reported by Lang, Fuchs, Denig, Maklakow, Franke, Purtscher,
Treacher Collins, Flemming ; they are often due to fracture involving
the infra-orbital nerve (Lang, Fuchs, Maklakow, Franke, Flemming).
The olfactory nerve has been affected (Lang), and the auditory (Talko,
Denig).

The deformity may increase during the early stages and is usually
permanent.

There has been much discussion as to the pathogenesis of the
condition.   Himly (1843) ascribed a preponderant *rôle* to displacement
of the pulley of the superior oblique.   More probable is the theory of
absorption of the orbital fat, which may be considered to be caused by
direct traumatism with pressure atrophy, or to atrophy from trophic
causes due to a primary nerve lesion.   This theory was accepted by
Nieden, but the period after the injury is often too short to account
for enophthalmos from this cause (Berlin).   More support has been
given to the theory of traumatic lesion of the sympathetic nerve (Talko,
Schapringer, Beer, de Schweinitz, Maklakow, Franke, Fischer,
Purtscher, Rohmer, Denig, Micas, Bistis, Panas, Luniewski).   As a
matter of fact there is very little to be said in favour of the theory, for
none of the known effects of stimulation nor of paralysis of the
sympathetic affords a reasonable explanation.   The enophthalmos of
paralysis of the sympathetic is very slight and transitory, and any
trophic effects which the nerve is supposed to exert are purely theo-
retical.   Careful collation of the pupillary conditions in published cases
affords no support to the theory, and in several cases directly negatives
it, *e. g.* Flemming, Cause.

Denig advanced the theory that the cause lay in lesion of the fifth
nerve.   That branches of the trigeminal are often affected has already
been mentioned, and enophthalmos sometimes occurs in trigeminal
neuralgia.   It is more reasonable to ascribe a trophic influence over
the orbital contents to the fifth nerve than to the sympathetic, but
there are many cases in which no considerable lesion of the trigeminal
can be proved.

The mechanical theories afford the best explanation.   For the most
part they imply a passive falling back of the eye owing to relative or
actual increase in the size of the orbit, or an active retraction due to

adhesions or muscular activity (Meltzer). Absolute increase in the orbital space by fracture and depression of the floor can scarcely be denied in the cases of Lang, Fuchs, Sachs, Franke, Manz, Flemming, Luniewski, and was proved by X rays in the cases of Chaillous and Birch-Hirschfeld. The depression theory was stated by Lang, and is supported by Fuchs, Ogilvy, Neulen, Franke, and Daulnoy; it is attacked by Beer, Denig, and Lederer, largely on account of misapprehension. There is no necessity to demand extension of the orbit directly backwards; any extension which will afford room for some of the orbital contents must be associated with falling back of the globe, and there is indeed often some depression (Lang, Beer, Cohn, de Schweinitz, Bruner, Shoemaker). On the other hand the depression theory cannot be made to explain all cases of traumatic enophthalmos. In some cicatricial shrinking of the retrobulbar tissues affords the best explanation (Gessner). Here again it is unnecessary to have recourse to inflammatory action, of which there is no clinical evidence in many cases. Mere trauma without excessive inflammatory reaction is quite sufficient for the purpose (Löw, Cohn, Ayres, Burnett, Treacher Collins, Praun). Lederer ascribes the formation of cicatricial bands to hæmorrhage. That such has probably occurred in some cases is shown by the optic atrophy in the cases of Talko, Löw, Beer, Schapringer, Ogilvy, Franke, Grunert, Birch-Hirschfeld, Purtscher, and others. Extensive orbital hæmorrhage, however, demands exophthalmos as an early sign, and this is often absent. In some cases the enophthalmos is of very early onset (Schapringer, Manz, Hansell, Purtscher, Flemming). Moreover, the theory demands very gradual onset, which renders it improbable as an explanation of some cases (Beer, Cause, Gessner, Denig, Kilburn, and Birch-Hirschfeld). At the same time it is likely that effusion of blood, however slight, is conducive to organisation and formation of fibrous tissue.

Birch-Hirschfeld has adduced another important factor, viz. laceration of the ligaments and fasciæ which support the globe, or fracture of the bones which form the attachments of these fasciæ. When these are loosened the tone of the recti will pull the globe backwards, and this tone is itself probably increased. The prevalence of injuries of the orbital margin down and in is striking in this connection. We must agree with Birch-Hirschfeld in holding that the factors which are at work in producing traumatic enophthalmos are various and manifold, and that the part which each plays varies in different cases.

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CONGENITAL ENOPHTHALMOS is usually associated with defective movements of the eye. Various degrees of the condition are by no means uncommon, though little attention has been paid to them until recently (Gulland, Treacher Collins, Türk, Inouye, Axenfeld and Schürenberg, Bergmeister, Walker). Birch-Hirschfeld has collected and collated 50 cases from the literature. Of these 30 were female, 20 male : the left eye was affected 35 times, the right only 10 ; both eyes are rarely affected (5 cases), and then the left is worse than the right. Hereditary influence is seen in the cases of Heuck, Best, and Wolff. Congenital defects in the eye occur—microphthalmia with coloboma of the choroid and optic nerve (Bergmeister) ; microphthalmia (Braunschweig, Nettleship) ; tortuous vessels (Spuler) ; pseudoneuritis (Best) ; hypermetropia, astigmatism, amblyopia (Schapringer, Evans, Knapp, Birch-Hirschfeld, Best, Bergmeister, Gradle). Facial asymmetry is noted by Bergmeister.

There may be parallelism of the axes, convergent or divergent strabismus in the primary position, without or with some enophthalmos (Weeks, Carpenter, Gradle, Bergmeister, Best, Inouye, Nettleship, Evans, Wolff, Schapringer, Axenfeld and Schürenberg, Spuler, Maclehose). Enophthalmos is nearly always most pronounced in adduction, sometimes in every attempt at movement (Hueck), or not increased by movement (Best, Bergmeister). The amount of retraction varies—Alling 10 mm., Wolff 8 mm., Axenfeld and Schurenberg and Inouye 5 mm., Birch-Hirschfeld 3 mm. Retraction is generally accompanied by narrowing of the palpebral aperture : the latter may be unassociated with retraction (Duane), or absent with retraction (Wolff). Abduction may be accompanied by definite exophthalmos and widening of the palpebral aperture (Spuler, Schapringer, Wolff).

The condition is not due to defect in the cerebral nuclei, as conjectured by Eason, nor will a lesion of the sympathetic nerve account for the signs. Anatomical investigation has proved the presence of abnormalities in the extrinsic muscles. Inouye found the external rectus replaced by a tendinous band, the hypertrophic internal rectus being inserted 6 mm. behind the limbus (cf. also Alling, Birch-Hirschfeld). Insertion of the internal rectus too far back was found by Heuck, Bahr, Axenfeld and Schürenberg, and Evans, and in several of these cases the external rectus was defective in muscle tissue. That in some cases posterior insertion of the internal rectus is alone necessary to produce the defect is shown by the fact that passive rotation inwards is accompanied by no retraction (Axenfeld and Schürenberg, Evans, Alling). Bahr and Knapp found a subsidiary insertion of the internal rectus anatomically, and this is conjectured by Axenfeld and Schürenberg for one of their cases : the band acts as a retractor bulbi. Parker, Peschel and Schapringer explain the retraction by contraction of the superior and inferior recti, which act as vicarious adductors and retractors (Parker). Protraction in abduction is ascribed to the obliques by Peschel and Spuler, but if this is true the effect

should be greatest when all the recti act defectively (Braunschweig, Best, Varese, Bergmeister), which is contrary to the fact. Treacher Collins and Varese point out that the offshoots of fascia from Tenon's capsule to the orbital walls oppose retraction by the muscles, which will occur if they are absent or inserted too far back. Axenfeld and Schürenberg consider that retraction is opposed rather by fascial strands from the muscle sheaths to the periosteum and the presence of a positive orbital pressure. There can be little doubt that most of the cases are efficiently explained by one or more of these peripheral factors, and that the malformations differ in individual cases.

Permanent enophthalmos may occur without increase on adduction or other movement (Best, Bergmeister). The presence of exophthalmos in abduction in several cases points to a muscular origin for the permanent enophthalmos. Insertion of the muscles, particularly the internal rectus, too far back affords the best explanation. In one of Bergmeister's cases all the recti were abnormally short or replaced by elastic bands. On the other hand in two of his cases there was permanent enophthalmos of 3—4 mm. with perfect movements of the eye: these can scarcely be due to muscular anomalies, but the accompanying facial asymmetry points to congenital malformation of the orbit or its retrobulbar contents, and the enophthalmos of extreme hypermetropia must be borne in mind. Moreover, injury at birth must be carefully eliminated (*v.* p. 1192).

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CHANGES IN THE POSITION OF THE EYE FROM NERVOUS CAUSES, ETC.

Exophthalmos of slight degree is caused by paralysis of the recti muscles owing to their loss of tone: it is naturally most evident in paraylsis of the third nerve on account of the number of muscles involved. It is also caused by irritation of the cervical sympathetic nerve owing to contraction of unstriped muscle-fibres in the orbit (Eckhard, Edmunds). The effect is much more pronounced in lower mammals in which the retractor musculature of the eye is more developed. In man the proptosis is very slight, and in all cases the vaso-constrictor effect of the sympathetic fibres is opposed to it.

Exophthalmos from *drugs* is probably generally due to stimulation of the sympathetic. This is almost certainly the case with cocain (Edmunds, Lewin and Guillery), the effect being enhanced, though

only apparently, by the retraction of the lids from contraction of Müller's muscles. It is well known that large doses of thyroid extract may produce exophthalmos. In rabbits 0·1 grm. by the mouth is sufficient (Cunningham), and the effect can be produced in monkeys (Edmunds). It has been observed in man by Béclère and Lawford. Inouye was unable to produce it in dogs. Paraphenylene diamine, a drug used for the hair, produces eczema, conjunctival irritation, and exophthalmos. Experiments on animals tend to show that the proptosis is due to œdema of the orbital tissues rather than to nervous agency (Matsumoto, Puppe, Kunkel, Pollak, Grunert). Exophthalmos associated with ptosis and paralysis of the third nerve has been recorded by Bach in lead poisoning.

Enophthalmos of slight amount is produced by paralysis of the cervical sympathetic. Most of the cases observed have been those in which the superior cervical ganglion has been removed (Zimmermann, Jacobsohn, Matlakowski). It is not mentioned by Weir-Mitchell, Morehouse and Keen, Seeligmüller, Chavasse, Bannister, Remak.

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CHAPTER XXIV

PANOPHTHALMITIS

THE histological changes which occur in panophthalmitis have already been described under the appropriate anatomical headings (see Vols. I and II).

Exogenous panophthalmitis.—The condition is usually due to exogenous infection of the vitreous by perforating wounds, and only when pyogenic inflammation has extended to the vitreous and its bounding membranes is the term suitable. Hence the progress of the inflammatory process through the vitreous and to the ciliary body, retina, and choroid is of prime importance. The studies of Fuchs on this subject have already been mentioned (Vol. II, pp. 447, 597). He has shown that the inflammation spreads first to the pars ciliaris retinæ and to the retina proper, so that intense suppurative retinitis is set up. Extension to the deeper parts occurs only where the retina is in close relationship with the uvea. The inner layers of the ciliary body therefore suffer early, whilst the choroid is affected only if the retina continues in apposition with it. In many cases the retina becomes detached at an early stage. The choroid is then attacked only by continuity from the ciliary body, and the spread is slow and localised. The optic disc may be affected early, and similar extension by continuity occurs into the posterior part of the choroid. An exception is found in those cases in which the supra-choroidal space is directly infected by the wound, when the inflammation spreads rapidly throughout it and to the choroid in its whole extent. Fuchs has found that the bacteria which set up the inflammation appear not to travel as fast as the inflammatory process. In early cases in which the inflammation is restricted to the anterior part of the vitreous, and even when bacteria lie deep in the vitreous, they are not found in immediate proximity to the retina. The retinitis and choroiditis are therefore set up by the toxins which the bacteria produce.

Excision of a suppurating eye may be followed by meningitis and death (v. Graefe, Deutschmann, Nettleship, and others).

v. GRAEFE.—K. M. f. A., i, 1863. DEUTSCHMANN.—A. f. O., xxi, 4, 1875. NETTLE-SHIP.—T. O. S., vi, 1886. MARSHALL.—R. L. O. H. Rep., xiv, 2, 1896. ENSLIN AND KUWAHARA.—A. f. A., l, 1904.

Bacteriology.—Herrnheiser has shown that vitreous humour, sterilised

by heat, is a worse culture medium for most bacteria than bouillon ; the exceptions are diptheria bacilli, cholera bacilli, and *B. pyocyaneus*. This is not true of the vitreous in the living eye.   Minute doses of typhoid or cholera bacilli cause not only panophthalmitis, but frequently general infection and death (Herrnheiser, Axenfeld, Gillet de Grandmont and Gasparrini).   *B. prodigiosus* and other saprophytes cause purulent inflammation in the vitreous (Sattler).

Bacillus subtilis, which is almost inert in the cornea and causes little reaction in the anterior chamber, causes intense suppuration in the vitreous.   Paplawska, working under Haab, found a bacillus— Haab's panophthalmitis bacillus—in a case of intra-ocular foreign body. Römer found a similar bacillus in a case of panophthalmitis after cataract extraction, and considered that it belonged to the subtilis group : this was confirmed by Silberschmidt and Kayser.   The types of hay bacillus occurring in the earth and their effect on the eye were studied by Polatti and Stregulina.

Bacillus subtilis is a motile, Gram-positive bacillus, $1\cdot2-3\ \mu$ long, $0\cdot8-1\cdot2\ \mu$ broad, which forms oval spores and long chains and filaments. Most forms are aërobic.   They grow on most media, best at $22°—37°$, not at all at $0°$ or $50°$.

The pathogenic action on the eye varies greatly (Perles and Lobanow, Bänziger and Silberschmidt, and others).   Inside the eye the organism usually disappears quickly, *e. g.* in seven days (Kayser). No spore formation occurs in the vitreous, but it is possible that spores introduced into the eye may cause late infection (Hess), though there was no such latent period in Ulbrich's experiments.   The toxin is intra-cellular, since filtered cultures are innocuous (Kayser).   Bacilli virulent in the vitreous produce no effect in the cornea, but this is not an absolute rule (Michalski, Gourfein, zur Nedden [ulcus serpens]).

Panophthalmitis has been produced by many other saprophytes, but the experiments show great variation in toxicity, etc. (Gifford, Perles, Lobanow, Stöwer, and others).

HERRNHEISER.—Prager med. Woch., 1894.   AXENFELD.—A. f. O., xl, 1894.   GILLET DE GRANDMONT AND GASPARRINI.—Ann. di Ott., xxiv, 1895.   SATTLER.—B. d. o. G., 1885. HAAB.—Fortschritte der Med., 1891.   PAPLAWSKA.—A. f. A., xxii, 1890.   SILBERSCHMIDT.— Ann. de l'Institut Pasteur, xvii, 1903.   KAYSER.—C. f. Bacteriol., xxxiii, 1902.   BIETTI, POLATTI.—Ann. di Ott., xxxiv, 1905.   ULBRICH.—A. f. O., lviii, 1904.   MICHALSKI.—C. f. Bacteriol., xxxvi, 1904.   AXENFELD.—K. M. f. A., xliii, Beilageheft, 1905.   DERBY.—Am. Jl. of O., 1905.   PERLES.—Virchow's Archiv, cxl, 1895.   RÖMER.—B. d. o. G., 1901. SMITH.—A. of O., 1905, 1906.   ZUR NEDDEN.—A. f. A., lii, 1905.   GIFFORD.—A. f. A., xvi, 1886.   STÖWER.—A. f. O., xlviii, 1899.   KAMPHERSTEIN.—K. M. f. A., xli, 1903.   STREGULINA. —Z. f. Hyg., li, 1905.   *AXENFELD.—Die Bacteriologie in der Augenheilkunde, Jena, 1907.

Bacteriological examination of human eyes with post-operative panophthalmitis has given the following results : *Staphylococcus pyogenes aureus* (Leber, Sattler, Weeks, de Schweinitz), ozæna or pneumonia bacillus (Terson and Gabriélidès), diphtheria group or xerosis bacilli (Kastalska, Gourfein), pneumococci (Gasparrini, Ewetzki, Mündler, Oertzen, Kuhnt, Schirmer and Flatau, Johnston, Woods and Johnston, Taylor, Duclos [7 cases], Axenfeld [7 cases]), streptococci (Duclos).   Pneumococci are therefore much most common in panophthalmitis after operation, usually cataract extraction ; they probably

originate in dacryocystitis, conjunctivitis, putting instruments in the mouth, etc. Hotta inoculated rabbits' corneæ with saliva and obtained pneumococcic infection in one third of the cases.

In panophthalmitis following injury pneumococci are not so common : streptococcus (Gallenga, Deutschmann, de Schweinitz, Enslin and Kuwahara, Axenfeld [5 cases]), staphylococci (Leber, Sattler, Gallenga, Axenfeld), *Bacillus subtilis (vide supra)*, *Bacillus pyocyaneus* (Sattler, Hanke), *B. coli* (Randolph), *B. pyogenes Passet* (Monti, Scimeni, Gallenga), *B. perfringens* (Chaillous), pneumococci (Gasparrini, Cuénod, de Lapersonne and Painblau, Mayweg, Lagrange, Axenfeld). Of course the cases which begin with hypopyon ulcer are mostly due to pneumococci, as well as those with adherent leucoma (Wagenmann, Terson, Dolganow and Sokolow).

Sattler.—B. d. o. G., 1889, 1892. Weeks.—Ophth. Rec., 1903. Terson and Gabriélidès.—A. d'O., xiv, 1894. de Schweinitz.—Ophth. Rev., xv, 1896. Mündler.—Ziegler's Beiträge, xxii, 1897. Oertzen.—K. M. f. A., xxxvii, 1899. Kuhnt.—Z. f. A., i, 1899. Schirmer and Flatau.—Z. f. A., ix, 1903. Schmidt and Hirota.—Z. f. A., vii, 1902. Woods and Johnston.—Ophth. Rec., 1904. Duclos.—Ann. d'Oc., cxxxiv, 1905. Hotta.—K. M. f. A., xliii, 1905. Hanke.—Z. f. A., x, 1904. McNab.—K. M. f. A., xliii, 1905. Randolph.—Am. Jl. of Med. Sc., 1893. Chaillous.—Ann. d'Oc., cxxxiv, 1905; Rec d'O., 1906. de Lapersonne and Painblau.—Rev. gén. d'O., 1897. Lagrange.—Rec. d'O., 1901. Wagenmann.—A. f. O., xxxv, 4, 1889. Terson.—Ann. d'Oe., cix, 1898. Dolganow and Sokolow.—A. f. A., xlvii, 1903. Flatau.—Z. f. A., ix, 1903. Johnston.—Med. News, 1904. *Axenfeld.—Die Bacteriologie in der Augenheilkunde, Jena, 1907.

Endogenous panophthalmitis (*metastatic ophthalmia*).—Metastatic purulent choroiditis and retinitis have already been discussed briefly (Vol. II, pp. 448, 598). The condition as a sequel of puerperal fever has been known since the time of Tenon (1775). Jüngken (1836) described hypopyon occurring in this disease as milk in the anterior chamber. The subject was treated by Fischer and v. Arlt, the first anatomical observations coming from Meckel (1854) and H. Müller (1856). Virchow first proved the existence of capillary embolism (1856).

Metastatic ophthalmia is most common as the result of infection from the female generative organs, especially in cases of puerperal fever. Mitvalsky records a case of gangrene of all four lids from septic embolism due to endometritis. Cases of iritis and iridochoroiditis are fairly frequent in the literature (v. Michel, Despagnet, de Wecker, Axenfeld, Bayer, Berger and Loewy, Foerster, Valude, Terrien, Veillon and Morax) ; in most of the cases there was endometritis. In puerpural fever retinitis septica and metastatic endophthalmitis were at one time common. Retinitis septica (Roth, *v.* Vol. II, p. 599), may occur in one or both eyes; it may start soon after parturition, *e. g.* five days (Gimurto), or late, *e. g.* twenty-three days (Berger and Loewy). The prognosis for life is bad (Roth, Litten), since septic endocarditis is not infrequently present ; Herrnheiser, however, found death in only five out of sixteen cases. Roth, Ischreyt, and Herrnheiser attribute the disease to toxins ; Litten, Leber, Kahler, and Wagenmann to bacterial embolism. Axenfeld thinks it may be due to toxins or exceptionally to pyogenic organisms of attenuated virulence. Metastatic endophthalmitis

develops rapidly with pain and usually loss of sight within twenty-four hours. The retinal veins are dilated; there are white spots and hæmorrhages in the retina and vitreous opacities. The white spots rapidly increase in size and ophthalmoscopic examination becomes impossible. Detachment of the retina may occur early (Hirschberg). Iritis, hypopyon, swelling of the lids, etc., supervene. Purulent panophthalmitis usually follows. According to Schmidt-Rimpler the disease is generally caused by bacterial emboli in the retinal vessels, often following malignant endocarditis. A case commencing with subconjunctival metastatic (?) abscesses is recorded by Feuer. Axenfeld found evidence of sympathetic ophthalmia following phthisis bulbi in these cases in 1·3 per cent. (Scheffels, Deutschmann, Walter and

FIG. 810.—METASTATIC ENDOPHTHALMITIS.
W. T. Holmes Spicer, T. O. S., xxvii. Section through abscess at the disc, showing colony of staphylococci.

Schwarzschild). The patients are usually multiparæ (78 per cent.), the youngest being twenty-five years old. The disease occurs in the second or third week, the latest recorded being in the seventh week. In sixty-nine cases one eye was affected in forty-two, both in twenty-seven; both may be affected simultaneously or less commonly at an interval (2—24 days). Many patients die in 5—58 days, or a mean of nine days after the onset of the ophthalmia. Bilateral cases may survive (Hirschberg and Henius, Januskiewicz, Kater, Fischer). In the unilateral cases death occurred in 66 per cent. (Axenfeld), 58 per cent. (Groenouw), endocarditis being present in more than half; the patient may live in spite of endocarditis (Kriz). Non-septic embolism of the central artery in puerpural fever is reported by Snell. Orbital abscess is recorded by Rosas and Strzeminski.

Metastatic ophthalmia is much less frequent in surgical pyæmia; Groenouw has collected sixty cases, twenty-six of which were after wounds or operations. More than half the patients were between twenty and fifty, a quarter over fifty; 79 per cent. were male. The ophthalmia commenced after 1—45 days; of fifty-three cases sixteen were bilateral, thirty-seven unilateral, and of the former 75 per cent. died, of the latter 54 per cent. In the four bilateral cases (Mackenzie, Beck, Leber, Goller) the eyes were affected simultaneously and went on to panophthalmitis.

Groenouw has collected thirty cases of cryptogenetic pyæmia (Leube) with metastatic ophthalmia. There is fever of a septic character and often endocarditis, but the site of entry of the pathogenic
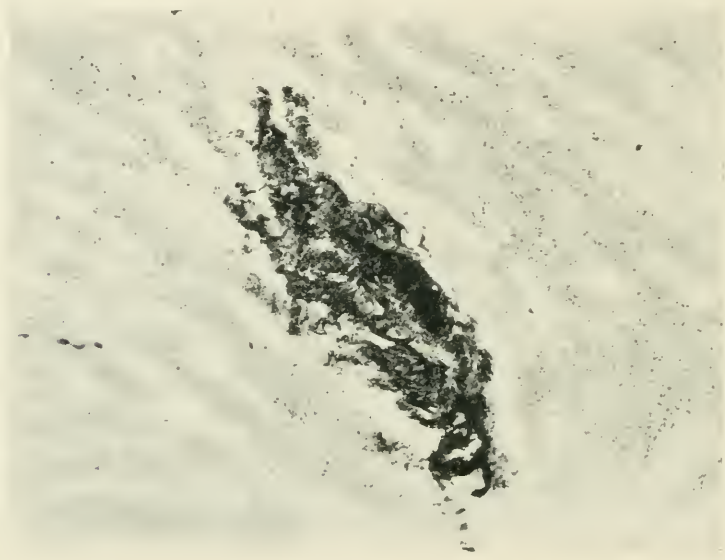
FIG. 811.—METASTATIC ENDOPHTHALMITIS.
From the same specimen, showing colony of staphylococci under higher magnification.

organisms cannot be discovered: in many cases it is probably in the gastro-intestinal tract. Half the cases were less than two years old, and 57 per cent. were male. In nineteen cases there was panophthalmitis in thirteen, phthisis bulbi without panophthalmitis in six. Of twenty-seven cases twelve were bilateral, fifteen unilateral: of the former 83 per cent. died, of the latter 33 per cent.

Metastatic ophthalmia also occurs in the course of specific infectious disease (q. v.).

The considerable amount of attention directed to metastatic inflammation of recent years emphasises the fact that other obscure subacute or chronic inflammations of the eye are probably due to this cause as well as the better recognised pyæmic suppurative conditions. It has been the custom in many of these to hypothecate a

toxæmia, the local manifestations being caused by the action of circulating toxins, the organisms which produce them remaining in some distant part of the body. It is highly probable that this view is correct in many cases, but it must be remembered that it is a mere conjecture, and there is little anatomical or experimental foundation for it. It is, of course, not sufficient to prove the absence of organisms from the eye, for it is well known that these may rapidly disappear whilst the deleterious consequences progress. On the other hand it is now quite definitely proved that many non-suppurative inflammations (*i. e.* non-suppurative in the generally accepted sense of the term) are due to metastatic inflammation caused by organisms circulating in the
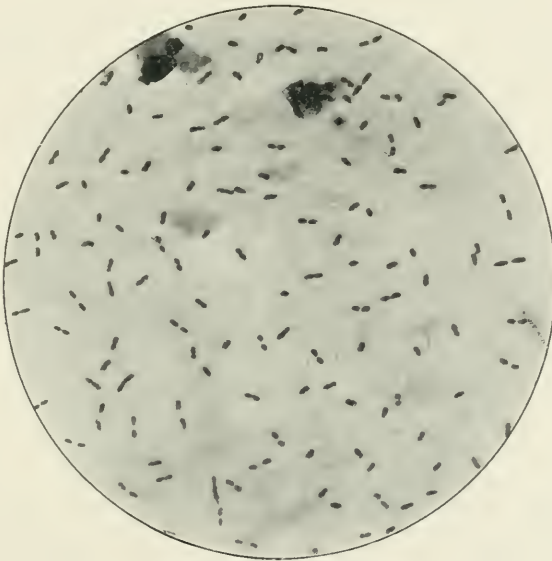
FIG. 812.—METASTATIC ENDOPHTHALMITIS.

Mayou, R. L. O. H. Rep., xvi, Case 1. From a photograph by Henderson. Smear preparation of pus from anterior chamber, stained by carbol fuchsin, showing *Diplococcus meningitidis*.

blood and forming emboli in the ocular capillaries. Subacute or chronic panophthalmitis of this type may subside without destruction of the eye. Cases occur in which one eye is lost by suppurative panophthalmitis whilst the other is attacked by a milder bacterial metastasis. The observations of Axenfeld and Goh are particularly important, for in such a case, which led to death, inflammatory nodules in the choroid and retina of the less affected eye contained masses of pneumococci. The organisms showed degenerative changes, and in other retinal patches had disappeared. Probably some cases of retinitis septica (*v.* Vol. II, p. 599) are due to bacterial metastasis, and Grunert and v. Michel have indeed found masses of streptococci. Similar choroidal patches have been seen in pneumonia (Fränkel, Peters). Cases of staphylococcic infection leading to changes in the
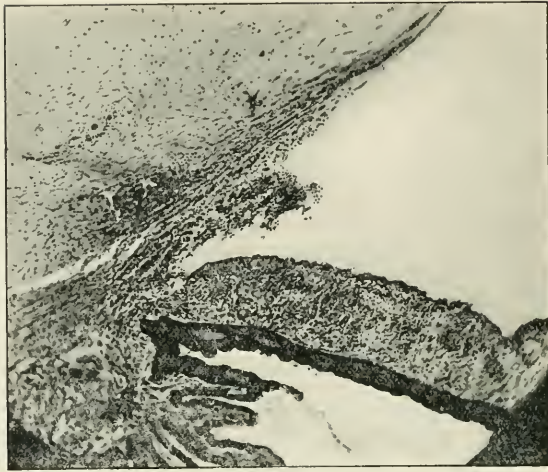
FIG. 813.—METASTATIC ENDOPHTHALMITIS.

From the same case, showing lower angle of anterior chamber. Note poly-
morphonuclear leucocytes in angle and around canal of Sclemm. There was pus
near the ora serrata, but the posterior part of the globe was normal.
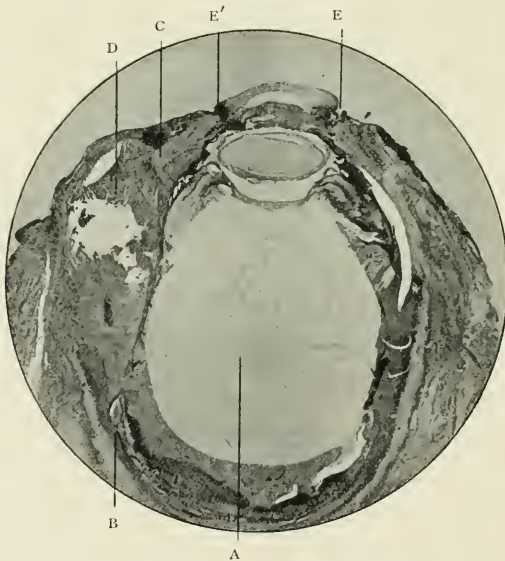
FIG. 814.—METASTATIC ENDOPHTHALMITIS.

Mayou, R. L. O. H. Rep., xvi, Case 2. Horizontal section. The vitreous, A,
was filled with pus, not shown owing to shrinkage by alcohol. The choroidal
vessels, B, are enormously dilated at the posterior pole. The pus has passed
through the choroid and sclerotic, C, and has formed an abscess beneath the tendon
of the internal rectus, D. The anterior chamber is filled with fibrinous exudate in
which there are few pus cells, E, E'. The ring ulcer of the cornea has perforated on
the outer side.

fundus with spontaneous resolution are reported by Holmes Spicer and Schanz. Probably a case which I saw and which has been published by Pernet comes under the same category. The woman, æt. 28, had acquired syphilis; during the secondary stage she complained of bad sight. The retinæ were œdematous and the vitreous of each eye full of exudates; there was a yellowish reflex, and an hypopyon appeared in the right eye. After prolonged treatment with anti-syphilitic remedies the vision in each eye became almost normal. In this case it is certain that the ocular inflammation was endogenous, and it is unlikely that it was due to syphilis uncomplicated by a septic process.

Sattler records the presence of Gram-positive cocci in a case of
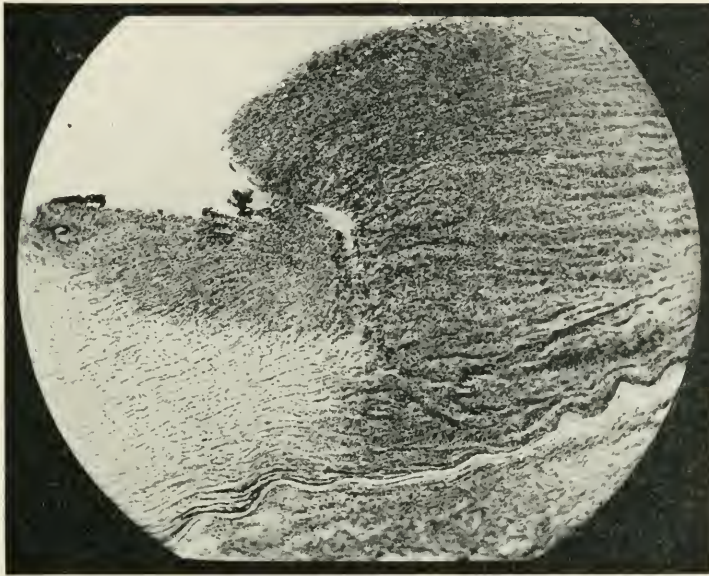
FIG. 815.—METASTATIC ENDOPHTHALMITIS.
From the same specimen. Section through the ring ulcer towards the inner side (Fig. 817, E′), showing the necrotic cornea comparatively free from leucocytes. Descemet's membrane is still intact.

plastic iridocyclitis. Fuchs, Gasparrini, and Schwartz found pneumo-cocci in purulent tenonitis.

Much light is thrown on embolic inflammation with bacteria by the experiments of Stock and Solenkowski and Woyzechowski. Stock obtained uveal inflammation in rabbits by intra-venous injection of *Bacillus pyocyaneus*. It is interesting to note that previous injury to the eye in rabbits and cats by cauterisation, foreign body, etc., increases the tendency to metastatic deposits with organisms of slight virulence, though these are easily obtained without such injury by intra-venous injection of virulent cultures of pyocyaneus, staphylococci, or streptococci (Woyzechowski).

With regard to metastatic panophthalmitis in puerperal cases

Axenfeld suggests that the streptococci of puerperal fever may have some specific affinity for the eye, for the eye was much less commonly involved in surgical pyæmia. Pneumococci, which were seldom the cause in either of these groups of cases, are much more frequent in the more obscure or cryptogenetic cases, but typhoid bacilli (Gasparrini, *cf.* Stock), bacterium coli (Loeser), pneumonia bacilli (Wopfner), etc., have also been found. Bertozzi found *Bacillus fusiformis* in Vincent's angina after measles. Subacute endogenous panophthalmitis may occur in tuberculosis (de Lieto Vollaro, Münch, Demaria). Several organisms have been held responsible in cases occurring after influenza—influenza bacillus (Tanja, Dianoux and Casali, Axenfeld [?]), *Staphylococcus aureus* (Eversbusch), staphylococci and streptococci (Despagnet), pneumococci (Haushalter and Vilber, Alfieri).

Bacterial embolism is the probable cause in metastasis after gonorrhœa and recurrent fever (Axenfeld). Poynton and Paine have cultivated a coccus (? *Streptococcus* or *Diplococcus rheumaticus*) from the blood in rheumatic fever which caused iridocyclitis when injected into a rabbit. Boucheron's good results with Marmorek's streptococcic serum in fifty cases of rheumatism points to endogenous metastasis of some kind. L. Müller has described miliary actinomycosis of the choroid.

JÜNGKEN.—Augenkrankheiten, Berlin, 1836. v. ARLT.—Krankheiten des Auges, Wien, 1853. MACKENZIE.—Diseases of the Eye, London, 1854. MECKEL.—Charité Annalen, 1854. H. MÜLLER (1856).—Gesammelte Schriften, Leipzig, 1872. VIRCHOW.—A. f. path. Anat. u. Physiol., ix, x, 1856. MITVALSKY.—K. M. f. A., xxxi, 1893. AXENFELD.—A. f. O., xl, 3, 1894 (Bibliography). *BERGER AND LOEWY.— Ueber Augenerkrankungen bei Frauen, Wiesbaden, 1906. VALUDE.—Ann. d'Oc., cxiii, 1885. TERRIEN.—A. d'O., xix, 1899. VEILLON AND MORAX. —Ann. d'Oc., cxiii, 1885. ROTH.—Deutsche Z. f. Chir., i, 1873. LITTEN.—Z. f. klin. Med., 1880 ; Die ophth. Klinik, 1901. HERRNHEISER.—K. M. f. A., xxx, 1892. ISCHREYT.— A. f. A., xli, 1900. LEBER.—A. f. O., xxvi, 3, 1880. WAGENMANN.—A. f. O., xxxiii, 2, 1887. HIRSCHBERG.—C. f. A., vii, 1883. FEUER.—C. f. A., v, 1881. SNELL.—Ophth. Rev., 1886. SCHEFFELS.—K. M. f. A., xxxviii, 1890. DEUTSCHMANN.—B. z. A., i, 1893. WALTER AND SCHWARZSCHILD.—Am. Jl. of O., 1893. HIRSCHBERG AND HENIUS.—C. f. A., ix, 1885. JANUSKIEWICZ.—C. f. A., xx, 1896. FISCHER.—C. f. A., xxi, 1897. ROCKLIFFE.—T. O. S., x, 1890. HAAB.—Korrespondenzbl. f. Schweizer Aerzte, 1903. DE SCHWEINITZ.—Ophth. Rec., 1905. AXENFELD AND GOH.—A. f. O., xliii, 1, 1897. GRUNERT.—Dissertation, Tübingen, 1902. FRÄNKEL.—A. f. O., xlviii, 2, 1899. PETERS.—K. M. f. A., xxxix, 1901. HOLMES SPICER.—T. O. S., xxvii, 1907. SCHANZ.—Z. f. A., xvi, Beilageheft, 1906. PERNET.—Berliner klin. Woch., 1904. SATTLER.—In Axenfeld, B. d. o. G., 1906. FUCHS. —Wiener klin. Woch., 1890. GASPARRINI.—Ann. di Ott., xxiii, 1895. SCHWARTZ.—B. z. A., xxx, 1898. STOCK.—K. M. f. A., xli, 1903 (Bibliography). SELENKOWSKI AND WOYZECHOWSKI.—A. f. A., xlvii, 1903. GASPARRINI.—Ann. di Ott., xxiv, 1895. STOCK, WOPFNER, PAUL.—K. M. f. A., xliv, 1906. LOESER.—Z. f. A., viii, 1902. BERTOZZI.— Ann. di Ott., xxxvi, 1907. DEMARIA.—K. M. f. A., xliii, Beilageheft, 1905 (Bibliography). DIANOUX AND CASALI.—Ann. di Ott., xxxvi, 1907. AXENFELD.—A. f. O., xl, 3, 1894. POYNTON AND PAINE.—Ophthalmoscope, 1903. BOUCHERON.—Ann. d'Oc., cxvi, 1896. FUCHS.—A. f. O., lviii, 1904. L. MÜLLER.—K. M. f. A., xli, 1903. UHTHOFF.—B. d. o. G., 1905. ELSCHNIG.—K. M. f. A., xliii, 1905. GROYER.—Münch. med. Woch., 1905. *AXENFELD.—Die Bacteriologie in der Augenheilkunde, Jena, 1907.

Taking the chief organisms seriatim *streptococci* usually cause severe purulent panophthalmitis because they are generally very virulent strains. This is especially true of the puerperal cases, and extraordinary multiplication of the bacteria is seen, particularly in the vitreous, but also in the retinal capillaries (Wagenmann, Vossius,

Axenfeld). Post-mortem growth must be borne in mind (*v.* Vol. II, p. 448). Necrosis of the tissues is very marked, especially in the retina with its end arteries. In the uveal tract, with profuse anastomosis, localised masses are commoner.

Axenfeld.—A. f. O., xl, 3, 1894. Wagenmann.—B. d. o. G., 1896. Haab.—Korrespondenzbl. f. Schweizer Aerzte, 1903. v. Michel.—Z. f. A., vii, 1902. Veillon and Morax.—Ann. d'Oc., cxi, 1894. Chaillous.—K. M. f. A., xliii, 1905. Liebrecht.—K. M. f. A. xli, 1903. Onfray.—A. d'O., xxiv, 1904. Bull.—T. Am. O. S., 1901. de Schweinitz.—Ophth. Rec., 1904. Stock, Pagenstecher.—K. M. f. A., xliv, 1906. *Axenfeld.—Die Bacteriologie in der Augenheilkunde, Jena, 1907.

Staphylococci in endogenous panophthalmitis have been found less frequently. A good example of a choroido-retinal abscess containing zooglœa masses of *St. aureus* from a case of pyelonephritis is figured by Axenfeld. The action is more localised, but there is extensive necrosis in the neighbourhood. Sachsalber has recorded a case of metastatic staphylococcic purulent scleritis. Holmes Spicer has recorded a case of retinal abscess, one of retinal phlebitis, and one of local detachment of the retina due to staphylococcic metastasis from boils; also a case of retinal phlebitis and local keratitis profunda due to ptomaine poisoning.

Axenfeld.—Die Bacteriologie in der Augenheilkunde, Jena, 1907. Pooley.—Am. Jl. of O., 1895. Liebrecht.—K. M. f. A., xli, 1903. Schanz.—Münchener med. Woch., 1906. Sachsalber.—Wiener med. Woch., 1898. Holmes Spicer.—T. O. S., xxvii, 1907.

Pneumococci appear to pass through the vessel walls relatively quickly, and rapid multiplication within the capillaries is seldom seen. Leucocytosis is extremely well marked, and phagocytes and cocci are intimately mingled (*cf.* ulcus serpens and the absence of an infiltration ring). The leucocytosis is not commensurate with the severity of the infection, and pneumococcic metastases have a relatively mild course. Those occurring in pneumonia rarely lead to the worst form of suppurative panophthalmitis (*cf.* Bietti), though such cases are also met with. Suppurative retinitis, etc., occurring in pneumococcic meningitis are true metastases, for there is evidence that the organisms do not travel down the nerve sheath (Axenfeld): the same was true in a case of traumatic streptococcic meningitis investigated by de Lieto Vollaro. According to Axenfeld there is no proved case of transmission by the nerve sheath as suggested in certain cases (Saltini, Silcock, Treacher Collins, and others).

Herrnheiser and Axenfeld.—A. f. O., xl, 3, 1894. Axenfeld and Goh.—A. f. O., xliii, 1, 1897. Ferri.—Ann. di Ott., xxvi, 1897. Alfieri.—Arch. di Ott., iv, 1897. Malfi.—Arch. di Ott., vii, 1899. Ahlström.—La Clinique ophth., 1897. Schwartz.—B. z. A., xxx, 1898. Silcock, Treacher Collins.—T. O. S., xx, 1900. Bull.—T. Am. O. S., 1901. Bietti.—K. M. f. A., xli, 1903; Ann. di Ott., xxxii, 1903. Petit.—Ann. d'Oc., cxxvi, 1901. Purtscher.—C. f. A., xxvi, 1902. Römer.—K. M. f. A., xl, 1902. Casali.—K. M. f. A., xli, 1903. Weeks.—Ophth. Rec., 1903. Zobel.—Z. f. A., xi, 1904. Morax.—Ann. d'Oc., cxxxii, 1904. Axenfeld.—Monatsschrift f. Psychiatrie u. Nervenh., 1897; Die Bacteriologie in der Augenheilkunde, Jena, 1907.

The *meningococcus intracellularis* (Weichselbaum) has been found several times (Wintersteiner, Axenfeld). Uhthoff found isolated cocci in sections, whilst Heine's search was negative. It would seem that

the organism quickly disappears within the eye. Zimmermann and Brown-Pusey's case was investigated only after a year's interval. The ocular inflammation is metastatic, the nerve sheaths being normal in the cases of Rudnew, Oeller, and Uhthoff.

WINTERSTEINER.—Wiener klin. Woch., 1901.   AXENFELD.—In Haglund, K. M. f. A., xxxviii, 1900.   UHTHOFF.—B. d. o. G., 1905.   HEINE.—Berliner klin. Woch., 1905. ZIMMERMANN AND BROWN-PUSEY.—Ann. of O., xii, 1903.

It is interesting to note that organisms appear to have a special predilection for certain parts of the eye. There can be no doubt that the retina was primarily affected in many cases (Virchow, Kahler, Nagel, Heiberg, Roth, Mackel, Axenfeld, Mitvalsky, Wagenmann, Vossius, Herrnheiser, Stock). This is particularly so when both eyes are affected; in unilateral metastasis embolic infection of the uvea is frequent (Axenfeld). It is difficult to account for the prevalence of retinal infection. It is not due to special narrowness of the capillaries for the uveal capillaries are equally small (Sattler). Predilection is very marked in many cases of non-suppurative metastases, if, indeed, these cases are due to such a cause (cf. "Pseudoglioma"). It is possible that certain tissues have special affinities for certain organisms, or in the phraseology of Ehrlich that only there are the specific receptors present. Primary tubercle of the retina, for example, is almost unknown, whilst the same disease frequently attacks the uveal tract. In rabbits the predisposition of the vascular part of the retina is absent. Stock never obtained retinal metastases with pyocyaneus, always uveal. It is doubtful if Selenkowski and Woyzechowski obtained them, and possibly different animals, e. g. cat, react differently. It was noticeable in Stock's experiments with tubercle that virulent cultures oftener produced slight lesions in the choroid than in the iris and ciliary body, and that this was due to encapsulation of the bacilli. Stock found that the transference of a completely healed tubercular iris from a blood infection in a rabbit to a second rabbit produced severe tuberculosis. This experiment is particularly interesting as explaining late exacerbations and recurrences in man, and the danger which may be anticipated from encapsuled organisms.

The importance of recent researches on metastatic infection in the eye can scarcely be overestimated, and they are particularly illuminating in connection with those chronic inflammatory processes which are so often observed clinically and so seldom respond satisfactorily to treatment, e. g. iridocyclitis, many forms of choroiditis, sympathetic ophthalmia (q. v.), etc.

*AXENFELD.—Die Bacteriologie in der Augenheilkunde, Jena, 1907.

CHAPTER XXV

ORBITAL CELLULITIS AND THROMBOSIS; THROMBOSIS OF THE CAVERNOUS SINUS

Orbital cellulitis may arise from extension of inflammation from surrounding parts—the nose and its accessory sinuses, the mouth *via* the pterygoid plexus, the cranial cavity, &c.—or rarely from metastasis. In many cases there is a history of injury, but this is often so slight that it is doubtful if it has any causal relationship. The primary source of the inflammation is not infrequently obscure, and an exhaustive investigation of the mouth, pharynx, nose, sinuses, etc., should be made. Treacher Collins and Walker have recorded two cases in which there was necrosis of the orbital plate of the frontal bone and I have seen a similar case. In my case there had been a slight blow on the face but no signs of the injury could be detected. An orbital abscess was opened, bacteriological examination giving a pure culture of *Staphylococcus aureus*. The patient, a boy, progressed well for a fortnight, when he became drowsy, and bilateral optic neuritis was discovered. Exploration of the orbit failed to reveal pus, but the orbital plate of the frontal bone was found to be bare and eroded. The frontal bone was trephined above the orbital margin and a large frontal abscess evacuated : a pure culture of *Staphylococcus aureus* was again obtained from the pus. At the post-mortem examination quiescent otitis media of long standing was discovered on the same side : the venous sinuses were normal, and it is probable that the ear disease played no part in the ætiology of the acute condition. In cases of this nature it is likely that the intestinal tract is not infrequently the source of sepsis.

Suppuration in the accessory sinuses of the nose is doubtless more often the cause of orbital phlegmon than is suspected. Thrombosis of the orbital veins, meningitis with or without the formation of extra-dural or intra-cerebral abscess, periostitis, etc., are frequent complications. The frontal, ethmoidal, and sphenoidal sinuses may be involved separately or together. Extension to the orbit may be by way of venous channels or by necrosis of the intervening bony plate and periostitis (Horner, Knapp, Leber, Carver, Panas, Ziem, Berger, Hirsch, Kuhnt, Winckler, Seifert, Vossius, Axenfeld, Kümmel, Eversbusch, and others).

There are many reports of orbital cellulitis in connection with

carious teeth or following extraction of teeth (Samelsohn, Vossius, Burnett, Snell, Schneider; optic atrophy, Hermann; optic neuritis, Hirsch; optic atrophy, Hallauer, Guttmann, Wicherkiewicz, Giulini). I have had a case under my care in which an abscess in connection with an upper molar was followed by orbital cellulitis on the opposite side. The oral abscess was opened and the orbital cavity drained, vision and ocular movements being perfectly restored.

HORNER.—K. M. f. A., i, 1863. SAMELSOHN.—Berliner klin. Woch., 1877. KNAPP.— A. f. O., ix, 1880. LEBER.—A. f. O., xxvi, 3, 1880. CARVER.—Brit. Med. Jl., 1883. VOSSIUS. —A. f. O., xxx, 3, 1884. PAGENSTECHER.—A. f. A. xiii, 1884. BURNETT.—A. of O., xiv, 1885. HUTCHINSON.—Brit. Med. Jl., 1885. ZIEM.—Allg. med. Centralzeitung, 1887. TREACHER COLLINS AND WALKER.—R. L. O. H. Rep., 1889. SNELL.—T. O. S., x, 1890. SCHNEIDER. —Deutsche Monatsschr. f. Zahnheilk., 1890. PANAS.—A. d'O., x, 1890. ZIEM.—Virchow's Archiv, cxxvi, 1891; Monatsschrift f. Ohrenheilk., 1893. BERGER.—Rapports entre les Maladies des Yeux et celles du Nez, etc., Paris, 1892. HERMANN.—Allg. Wiener med. Zeitung, 1893. HIRSCH.—Wiener med. Woch., 1893; Prager med. Woch., 1893. LAWSON.—T. O. S., xv, 1895. KUHNT.—Ueber die entz. Erkrankungen der Stirnhöhlen, etc., 1895. HALLAUER. —A. f. A., xxxvii, 1898 (Bibliography). WINCKLER.—Ueber Beziehungen zwischen einigen Affektionem der Nase, etc., und Augenkrankheiten, 1898. SEIFERT.—Münchener med. Woch., 1898. DAGILAISKI.—K. M. f. A., xxxvii, 1899. GUTTMANN.—C. f. A., xxiii, 1899. VOSSIUS.—Z. f. A., iv, 1900. AXENFELD.—Deutsche med. Woch., 1902. KÜMMEL.— Bergmann's Handbuch d. prakt. Chir., 1903. EVERSBUSCH.—In G.-S., ix, 1903. WEISS.— Z. f. A., x, 1903. REIS.—A. f. O., lix, 1904. GIULINI.—Münchener med. Woch., 1904. KAISER.—A. f. O., lxi, 1905. BARTELS.—A. f. A., lvi, 1906. PAUNZ.—Z. f. A., xvi, 1906.

The *bacteriology* of orbital cellulitis is intimately concerned in the bacteriology of diseases of the surrounding parts. As regards the accessory sinuses of the nose it has been shown that pneumococci are most frequently found in empyemata : pneumococci plus streptococci, streptococci, and staphylococci are often found, influenza bacilli, diphtheria, and meningococci rarely. In the foetid cases many anaërobic saprophytes occur. The orbital cellulitis may progress after the sinus empyema has discharged (Axenfeld). As regards the organisms actually found in orbital cellulitis pneumococci have been reported by Hirsch, Kuhnt, Guignot and Cabannès, Faure, de Lapersonne, Weiss, Lefrançois, Axenfeld, Vossius, Thomson ; streptococci by Berger, Reis, Panas, Trousseau, Axenfeld, Brand, Hirsch, Thomson ; staphylococci by Pergens, Axenfeld, de Lapersonne, Brand, Thompson, Orlandini ; influenza bacilli by Siegrist, Axenfeld and Brand. Cellulitis occurring in pneumonia or influenza is not always caused by the pneumococcus or influenza bacillus respectively ; the depressed constitutional condition affords an opportunity to pre-existing bacteria, mostly derived from the nose in all probability.

Orbital cellulitis occurring in young children seldom arises from the accessory sinuses of the nose, since these are relatively undeveloped up to eight years of age. During the first few weeks (Dujardin, Leplat) a birth injury is the probable cause. In older children (Cabannès and Lamarque, Trousseau, Müller) extension from the lids or metastasis affords the most likely cause. The youngest case definitely due to sinus trouble (frontal sinus) was nine years old (Axenfeld). The contents of mucoceles of the accessory sinuses are usually sterile (Axenfeld), and this may be the case of the orbital pus (Hirsch), though very rarely.

There is no doubt that the orbital infection may lead to infiltration only without abscess formation (Axenfeld and Gruber). On the other hand temporary exophthalmos in such cases may be due merely to œdema.

Though rare, metastatic orbital cellulitis certainly occurs (Schwend, Axenfeld, Müller). In Müller's cases streptococci were found ; in two probable cases of Pergens pneumobacilli were found in one, *S. aureus* and *B. pyocyaneus* in the other ; Loeser found *B. coli*. Reis in a case of pustule in the upper lip (staphylococci and streptococci) found cellulitis and an abscess in the optic nerve at the lamina cribrosa ; a venous transmission was conjectured. Panas recorded an interesting case of angioma of the orbit electrolysed during typhoid fever ; orbital suppuration ensued and typhoid bacilli were recovered from the pus.

Primary osteomyelitis occurs in the walls of the orbit (v. Ammon). Tubercle shows a predilection for the malar bone, but pyogenic organisms also occur, *e. g.* staphylococci (Morax). Periosteal abscesses following injury are common, and the exact rationale offers considerable difficulty : they may be due to endogenous infection of a spot whose vitality has been lowered, or latent sinus trouble, etc., may be present ; such sinus trouble may also, however, be due to the trauma.

Purulent Tenonitis may certainly be endogenous in some cases (Fuchs, Mazza, Rollet, Bronner, Purtscher). Non-purulent Tenonitis is usually ascribed to the influence of toxins, but the possibility of attenuated bacterial metastasis, as in intra-ocular lesions (v. p. 1214), must be fully appreciated.

The danger of excision of the eye during suppurative panophthalmitis was insisted upon by v. Graefe. Enslin and Kuwahara traced cords of streptococci along the lymph spaces and optic nerve sheaths to the meninges in such a case where meningitis supervened ; macroscopically the orbital tissues were normal. Meningitis may occur after exenteration of the globe (Pes, staphylococci in the eye, streptococci in the meninges ; Bocci, pneumococci). In Alfieri's case the panophthalmitis was itself of pneumococcic endogenous origin. In meningitis following septic wounds of the eye de Lapersonne found pneumococci : he obtained meningitis experimentally by infecting the nerve sheath. That meningitis is rare clinically in panophthalmitis is probably due to obliteration of the channels by inflammatory coagula and thrombosis.

Facial erysipelas is a well-known cause of orbital cellulitis, with or without meningitis. The streptococci travel along the lymph sheaths of the nerves and vessels, the vaginal space of the optic nerve, and the veins (thrombophlebitis) (Leber, Addario, Bartels).

Meningitis may give rise to orbital cellulitis by direct transmission. It is a rare cause (Axenfeld, de Lieto Vollaro), and panophthalmitis has not been proved due to it. Hoffman found an abscess in the optic nerve sheath in a case of meningitis. Weeks reported a case of orbital cellulitis following abscess of the frontal lobe. On the other hand I have seen a case of orbital abscess with necrosis of the orbital plate of the frontal bone in which a frontal abscess developed at a late stage.

Orbital cellulitis from direct injury or infectious processes in the immediate neighbourhood (Laas and others) is relatively common.

No sharp line can usually be drawn between orbital cellulitis and thrombosis of the veins in most of these cases.

Cases of actinomycosis of the orbit have been reported by Partsch, Ransom, Darier and Gauthier, Coppez and Depage, Vossius, Koch, Axenfeld, Weeks.

STANCULÉANU AND BAUPP.—Internat. Med. Congress, Paris, 1903. ZARNIKO.—Handb. d. Erkrankungen d. Nase, Berlin, 1905. EVERSBUSCH.—In G.-S., ix, 1903. BRONS.— Lubarch and Oestertag's Ergebnisse, 1900–1905. AXENFELD.—Deutsche med. Woch., 1902. HIRSCH.—Prager med. Woch., 1894. KUHNT.—Die Erkrankungen der Stirnhöhle, 1895. FAURE.—Thèse de Bordeaux, 1905. DE LAPERSONNE.—Congrès franç. d'O., 1903. WEISS.— Z. f. A., x, 1904. VOSSIUS.—Z. f. A., iv, 1900. THOMSON.—Brit. Med. Jl., 1906. PANAS.— A. d'O., xv, 1895. BRAND.—Dissertation, Freiburg, 1902. SIEGRIST.—A. f. O., xl, 3 and 4, 1894. DUJARDIN.—Jl. des Sc. méd. de Lille, 1888. ORLANDINI.—La Clin. oc., 1904. TROUSSEAU.—Ann. d'Oc., cxix, 1898. MÜLLER.—Dissertation, Würzburg, 1905. AXENFELD. —A. f. O., xl, 3, 1894. PERGENS.—Ann. d'Oc., civ, 1895. LOESER.—Z. f. A., viii, 1902. REIS. —A. f. O., lix, 1905. V. AMMON.—A. f. A., xlix, 1904. MORAX.—Soc. franç. d'O., 1905; Rec. d'O., 1905. ROLLET.—Ann. d'Oc., cxxviii, 1902. BRONNER.—T. O. S., xxiv, 1904. PURTSCHER.—C. f. A., xxviii, 1904. ENSLIN AND KUWAHARA.—A. f. A., l, 1904 (Biblio-graphy). PES.—K. M. f. A., xliii, 1905. BOCCI, ALFIERI.—Arch. di Ott., iv, 1896. LEBER. —A. f. O., xxvi, 3, 1880. ADDARIO.—Arch. di Ott., xii, 1904. BARTELS.—A. f. A., lvi, 1906. AXENFELD.—A. f. O., xl, 3, 1894; Monatsschr. f. Psych. u. Nervenh., 1897. DE LIETO VOLLARO.—K. M. f. A., xli, 1903, Beilageheft. LAAS.—Z. f. A., vii, 1902. PARTSCH.—C. f. A., xvii, 1893. RANSOM.—Brit. Med. Jl., 1896. DARIER AND GAUTHIER. —Jl. méd. de Bruxelles, 1902. COPPEZ AND DEPAGE.—Jl. méd. de Bruxelles, 1903. VOSSIUS. —B. d. o. G., 1902. KOCH.—Ophth. Klinik, 1904. AXENFELD.—Deutsche med. Woch., 1902. WEEKS.—New York Eye and Ear Infirmary Rep., 1897.

Thrombosis of the cavernous sinus may arise from extension from the ophthalmic veins, the source of infection being wounds or septic infections of the skin, etc., or from the ear, nose and accessory sinuses, pharynx, tonsils, etc., or as a metastasis in infectious diseases or septic conditions.

The anatomy of the venous channels which communicate with the cavernous sinus is of prime importance for the comprehension of thrombosis affecting it. The superior and inferior ophthalmic veins enter it in front and the superior and inferior petrosal sinuses leave it behind. It communicates directly with the pterygoid plexus through the middle meningeal veins and the vein of Vesalius, and indirectly through a communicating vein from the inferior ophthalmic to the pterygoid plexus. The anastomoses of the ophthalmic veins with the frontal and angular open up a communication with the face. Labyrinthine veins opening into the inferior petrosal sinus afford a communication with the middle ear. Numerous tributaries throw it into direct or indirect communication with most parts of the cerebrum. The mastoid emissary vein places the sinus in communication with the subcutaneous tissues behind the ear through the sigmoid sinus and superior petrosal sinus: it is this communication which is of great diagnostic importance, since swelling behind the ear may decide the question of thrombosis of the cavernous sinus. The absence of valves in most of these communicating branches facilitates the spread of thrombosis in each direction along them. The sinus of one side communicates with that of the other by two (or sometimes three) transverse sinuses which surround the pituitary body. Hence the transference of thrombosis from one side to the other is easy, and frequently marks the last stages of the case.

The first sign of such transference is paralysis of the sixth nerve of the opposite side with defective abduction of the eye, and this sign should be carefully watched for in any suspicious case of inflammatory unilateral exophthalmos.   The early implication of the sixth nerve is due to its position in the cavernous sinus, and the relationship of the nerves which pass through the sinus to each other and to other important structures should be borne in mind.

Pathological changes occur in the fundus in about 20 per cent. of cases, other than otitic or traumatic (Uhthoff).   Optic neuritis is reported by Schmidt, Pflüger, Ellett, Macewen, and others, choked disc by Wernicke, Paunz, Bartels, Kampherstein and others ; optic atrophy by Leber and others.   When the orbital veins are not involved ophthalmoscopic signs and exophthalmos are likely to be absent. Pronounced papillitis is almost always indicative of extensive implication of the orbital veins and tissues, though it may be due to intracranial abscess.   Bilateral exophthalmos and papillitis occur especially in sinus thrombosis following disease of the sphenoid sinuses (Kander, Bronner, StClair Thomson and others).

It is commonly stated that thrombosis of the cavernous sinus causes dilatation of the retinal veins (Bronner, Ellett, Macewen, and others). This is certainly not so in many cases, and its absence is readily explained by the free anterior anastomoses of the ophthalmic veins and the posterior communications with the pterygoid plexus.   When it occurs unusually extensive thrombosis of the veins of the orbit must be predicated.

Metastatic purulent ophthalmia is rare (*cf.* Schmidt).   Leber considers that thrombosis of the central retinal vein accounts for great loss of vision with comparatively slight orbital signs, a view supported by observations of Angelucci and v. Michel, and by anatomical proof in the case reported by Mitvalsky.   Bartels found circumscribed thromboses in the retrobulbar part of the central artery and vein, without marked signs in the fundus.   These observations require confirmation in the light of recent research on thrombosis of the central vein (q. v.).

Exophthalmos in cases other than otitic or traumatic is the most constant sign, occurring in 72 per cent. of cases (Uhthoff).   Consecutive transference from one sinus to the other occurs in 50 per cent. of cases (Macewen).   Simultaneous implication of both cavernous sinuses occurs in suppurative affection of the sphenoid (Jack, Raymond, Bartels), though it may be consecutive (Bronner, Ellett, Reis) or remain unilateral (Jessop, Onodi, Kander, Schröder, Finlay).

Thrombosis of the cerebral sinuses of otitic origin causes typical choked disc in 18 per cent. of cases (Uhthoff), whilst pathological changes in the fundus are seen in 54 per cent.   Only 25 per cent. of the cases with choked disc can be regarded as uncomplicated, 33 per cent. having meningitis, 20 per cent. subdural abscess, 15 per cent. cerebral abscess, 7 per cent. cerebral abscess and arachnitis.   The papillitis often commences on the side of the lesion and continues more pronounced on that side—a point of diagnostic value.   Uhthoff found papillitis of moderate degree, not definite choked disc, in 24 per cent. of cases, 30 per cent. complicated with meningitis, 12 per cent. with

meningitis and cerebral abscess, 2 per cent. with meningitis and pachymeningitis, 20 per cent. with cerebral abscess, 14 per cent. with extra-dural abscess, 22 per cent. without further intra-cranial complication. It is almost always bilateral. Hyperæmia of the papilla and venous congestion of the fundus occurred in 10 per cent. of Uhthoff's cases. Marked optic atrophy is rare (Voss and others), and is sometimes due to other causes (Brieger).

Exophthalmos is much less common in otitic cases of thrombosis of cerebral sinuses than in others (9 per cent. Uhthoff) : it probably always implies affection of the cavernous sinus and extension to the orbital veins.

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Bacteriology.—It is desirable that further research should be directed to the bacteriology of thrombosis of the orbital veins and cavernous sinus, for the results already available show incongruities which throw doubt upon their accuracy. Whilst Nonne, Cabannès, and Axenfeld found *Staphylococcus aureus* in the veins in cases due to furuncle, as might indeed be expected, Villard found streptococci in a similar case. So, too, Mitvalsky found *Staphylococcus aureus* in a case due to extension of erysipelas; in a case starting in the tonsils he found pneumococci and pneumobacilli. Further it is peculiar that a septic process commencing on one side, *c. g.* in one tonsil, may produce thrombosis of the opposite cavernous sinus (Leber, Panas, Terson).

GÜNSBERG, MITVALSKY.—A. d'O., xvi, 1896. HOFFMANN.—Z. f. A., xvi, Ergänzungs- heft, 1906. TERSON.—Rec. d'O., 1893. STOCKER.—A. f. A., xxxiv, 1901. VILLARD.— A. d'O., xv, 1895. MÜLLER.—Dissertation, Würzburg, 1905. NONNE.—Münchener med. Woch., 1904. CABANNÈS.—Jl. méd. de Bordeaux, 1904. ROCHON-DUVIGNEAUX AND ONFRAY.—Arch. de Méd. des Enfants, 1905. MORAX.—Soc. franç. d'O., 1905. BARTELS.— A. f. A., lvi, 1906. AXENFELD.—Die Bacteriologie in der Augenheilkunde, Jena, 1907.

CHAPTER XXVI

SYMPATHETIC OPHTHALMIA

SYMPATHETIC ophthalmia may be defined broadly as inflammation of the eye resulting from disorder originating in the other eye. The eye first involved is called the exciting (sympathisierende, sympathisant, simpatizzante) eye; the eye second involved is called the sympathising (sympathisierte, sympathisé, simpatizzato) eye; the difference in English and Continental nomenclature should be carefully observed.

The earliest reference to sympathetic ophthalmia is found in the oldest German text-book of ophthalmology by Bartisch (1583), who remarks that in cases of injury to one eye " the other good eye is besides also in great danger." Le Dran (1741) says " si comme aux abscès qui se font ailleurs on attend que le pus soit fait le malade pourra perdre la vue par l'inflammation qui se communiquera à l'autre œil le long du nerf optique." Two undoubted cases are recorded by Demours (1818). Wardrop (1819) reported a case of sympathetic iritis briefly, and mentioned that veterinary surgeons had noticed destruction of the second eye in horses, and that it might be avoided by destruction of the first eye by lime (*cf.* Barton, Crompton). Lawrence (1833) recorded several cases. The true history of sympathetic ophthalmia may be said, however, really to have commenced with the masterly description and imaginative insight of William Mackenzie (1835).

Sympathetic ophthalmia was first mentioned under this designation in Germany by v. Ammon (1838). Neither he nor Himly (1843) appear to have been conversant with Mackenzie's work; his name is not mentioned and the descriptions are much less accurate and complete.

The next notable contribution to our knowledge of the disease was made by Augustin Prichard (1851), of Bristol, who first proved the efficacy of excision of the exciting eye. This treatment was received with distrust, even by v. Graefe, who preferred iridectomy, and finding this useless, resorted to the production of panophthalmitis by passing a thread through the eye; he mentions optico-ciliary neurotomy, but does not appear to have employed it. v. Graefe later recognised the value of excision of the exciting eye, and advanced the opinion (1866) that inflammation of the uveal tract was *sine quâ non* to sympathetic inflammation. At the Ophthalmological Congress at Heidelberg in 1863 George Critchett contributed important additions to our knowledge of the clinical picture of the disease, v. Graefe distinguished between

the more malign plastic and less grave "serous" forms of sympathetic iritis, and Donders adduced evidence of a sympathetic neurosis which is promptly cured by excision of the exciting eye.

The history of the disease from this point deals chiefly with the remarkable diminution in its frequency resulting from improved methods of treating perforating wounds, dependent largely if not entirely upon anti- and a-sepsis, and with discussions and experiments directed to solve the mystery of its pathogenesis, matters which will be considered later.

BARTISCH V. KÖNIGSBRÜCK.—Ὀφθαλμοῖουλεία, Dresden, 1583. LE DRAN.—Traité ou Réflexions tirées de la Pratique sur les Plaies d'Armes à Feu, Amsterdam, 1696. DEMOURS. —Traité des Maladies des Yeux, Paris, 1818. WARDROP.—Morbid Anatomy of the Human Eye, London, 1819. *MACKENZIE.—Practical Treatise on the Diseases of the Eye, 2nd ed., London, 1835. LAWRENCE.—A Treatise on the Diseases of the Eye, London, 1833. BARTON.—Medical Gazette, 1835. CROMPTON.—Medical Gazette, 1837. v. AMMON.— De Iritide, Preisschrift, Leipzig, 1838. HIMLY.—Die Krankheiten u. Missbildungen d. menschl. Auges, Berlin, 1843. *PRICHARD.—Provincial Med. and Surg. Jl., 1851; Ann. d'Oc., xxxii, 1854. v. GRAEFE.—A. f. O., ii, 2, 1856; iii, 2, 1857; vi, 2, 1860; ix, 3, 1863; xii, 2, 1866. COOPER.—Wounds and Injuries of the Eye, London, 1859. CRITCHETT, B. d. o. G., 1863.

Sympathetic irritation.—It has long been customary to describe two forms of sympathetic affection, one a milder, less grave sympathetic "irritation," the other, accompanied by definite signs of ocular imflammation of grave import, sympathetic "inflammation" or ophthalmia. Whether the former is but a milder form or premonitory stage of the latter or is quite distinct has been much discussed, and cannot yet be said to be finally settled, nor, indeed, is it likely to be until the pathogenesis of sympathetic inflammation is elucidated.

The manifestations of sympathetic irritation are protean. There is pain in and around the eye in the domain of the fifth nerve. "Sympathetic amblyopia" is often described, though the meaning attached to the term varies extraordinarily in different reports. From a collation of twenty-five cases Schirmer describes it as being usually a moderate diminution of central vision, which may, however, reach a high degree. It sometimes shows variations which coincide with exacerbations and remissions in the exciting eye; exact estimation of the acuity of vision is difficult owing to the irritability of the eye. Sometimes there is periodic clouding of the whole field of vision, which may show concentric contraction. Rapid onset of fatigue is a conspicuous feature of the visual act (cf. Liebreich, Laqueur). Accompanying these symptoms are "photophobia," lacrymation, and paresis of accommodation, usually of earlier onset than the "amblyopia." Sometimes the photophobia comes on with the slightest cause, such as looking at white paper, more frequently only after using the eyes for a time. The exciting eye is always painful and irritable or inflamed, though definite proof of intra-ocular inflammation may be wanting; usually it is shrunken (phthisis bulbi). The interval after the original injury to the first eye is generally several years, but sometimes there has been a recent exacerbation of irritation in the exciting eye. Photophobia may be intense, as in Hirschberg's case.

Photopsiæ may be present, usually bright flashes, seldom colours

(Schweigger, Mooren). Cuignet described subjective phosphenes of long duration, and also very prolonged duration of after-images.

Paresis of accommodation is a common symptom. . Usually it increases rapidly during near work and passes off after rest. Convex glasses are of little use since retinal asthenopia is also present in most cases. Much rarer is spasm of the ciliary muscle. Spastic miosis is also rare (Pagenstecher). Spasm of the orbicularis and nystagmoid jerking (Cuignet) are associated with the photophobia.

Lacrymation, increased by light or near work, is a constant symptom.

Schirmer's analysis of twenty-nine cases of so-called " sympathetic glaucoma " leads him to deny its existence. If there is a predisposition to glaucoma its progress may be accelerated (Schirmer).

The eye is injected, the pericorneal vessels being sometimes most affected, sometimes the conjunctival.

As regards the ætiology of sympathetic irritation the exciting eye is usually in a condition of phthisis bulbi, but it must be remembered that symptoms indistinguishable from sympathetic irritation may result from the mere presence of a foreign body on the cornea, or from staphylomata, glaucoma, iritis, and many other causes. Enucleation of the exciting eye is not a positive cure for the condition, and it is in many cases associated with a badly fitting artificial eye, painful scars in the socket, etc. It has been recorded after Mules's operation and after incomplete removal of the globe. Schirmer considers that there is always a condition of irritation of the ciliary nerves; he therefore advocates optico-ciliary neurotomy as the therapeutic measure *par excellence*.

With regard to the relationship of sympathetic irritation to sympathetic ophthalmia Schirmer considers that if the former is a mild form or premonitory stage of the latter the following postulates should hold good : (1) all diseases of the exciting eye which give rise to one type should also give rise to the other : (2) every sympathetic irritation of long duration or great intensity should gradually pass into sympathetic inflammation ; (3) every sympathetic inflammation should be preceded by a stage of sympathetic irritation. It is obvious that these postulates do not hold good. In my opinion so-called sympathetic irritation is a symptom-complex of two-fold significance. In the majority of cases it is a purely reflex phenomenon differing in no fundamental manner from the reflex symptom-complex affecting the exciting eye. Just as a foreign body on one cornea causes closure of both eyes, so prolonged irritation of one eye, whether from a simple cause or from the *causa causans* of sympathetic ophthalmia, will induce reflex blepharospasm, lacrymation, and blurring of vision of both eyes. On the other hand the early signs of true sympathetic ophthalmia may—though they rarely do—simulate sympathetic irritation. I have examined eyes which have been removed because they were " dangerous," though the symptoms were those only of slight sympathetic irritation, and the condition of the exciting eye could only as the result of great clinical experience be recognised as actually dangerous. In such eyes I have sometimes been surprised to find

definite early signs of uveitis of the type which is common in sympa-thetic ophthalmia, *i. e.* chiefly aggregations of mononuclear lympho-cytes in the iris, ciliary body, and choroid (*v.* Vol. I, pp. 292, 353; Vol. II, p. 460).

Sympathetic irritation has been attributed to irritation of the ciliary nerves of the exciting eye. This view is held to be supported by the favourable effect of intra-ocular section of the ciliary nerves (Meyer, Secondi, Lawrence) or by optico-ciliary neurotomy. According to the ciliary nerve theory the corresponding nerves of the opposite eye act as efferent channels. This theory will receive further con-sideration in dealing with sympathetic inflammation. It is accepted by Schirmer, but it seems to me to fail entirely to explain satisfactorily the manifold symptoms of the condition. If the opinion which I have expressed above is correct most cases are examples of easily compre-hensible though complex reflex action. The remainder are cases of mild or initial stages of sympathetic inflammation and therefore have the same pathogenesis as that disease.

Out of 200 injured eyes examined anatomically by Fuchs sixteen had caused sympathetic irritation. In none of these eyes were there the characteristic signs of sympathetic inflammation (*v. infra*).

Cooper.—Wounds and Injuries of the Eye, London, 1859. Mooren.—Ueber sympa-thische Gesichtsstörungen, Berlin, 1869; Ophthalmologische Mittheilungen, Berlin, 1873. Vigneaux.— De l'Ophthalmie sympathique, Paris, 1877. Roosa.—New York Med. Rec., 1878. Landesberg.—K. M. f. A., xvii, 1879. Yvert.— Rec. d'O., 1879. Chisholm.— New York Med. Jl., xxxi, 1880. Rosenmeyer.—A. f. A., xxviii, 1893. Nuel.—A. d'O., xxvii, 1897. Greeff.—A. f. A., xxvi, 1893. Liebreich.—In Critchett, B. d. o. G., 1863. Laqueur.—Étude sur les Affections sympathiques de l'Œil, Paris, 1869. Hirschberg.— Klinische Beobachtungen, Wien, 1874. Schweigger.—A. f. A., xv, 1885. Cuignet.— Rec. d'O., 1878. Lindsay Johnson.—A. d'O., xii, 1892. Pagenstecher.—Klinische Beobachtungen, Wiesbaden, 1862. *Schirmer.—In G.-S., vi, 2, 1900 (Bibliography). Fuchs.—A. f. O., lxi, 1905.

Sympathetic inflammation.—Schirmer rightly points out that the chief difficulty in dealing with sympathetic ophthalmia is that it is always only a presumptive diagnosis; the symptom-complex is never pathognomonic, and it is of the utmost importance to bear this in mind in reviewing the enormous literature which has accumulated upon the subject.

It is difficult to obtain accurate statistics as to the *frequency* of the disease. One fact alone stands out prominently, viz. that it has diminished greatly in frequency since the introduction of anti- and a-septic modes of treatment of perforating wounds and under the improved technique of modern methods, in spite, too, of greater con-servatism. Mooren records 146 cases in 108,416 eye patients from 1856 to 1881, or 0·134 per cent.; O. Becker 18 in 12,365 in-patients from 1868 to 1888, or 0·15 per cent. Treacher Collins gives the following statistics for Moorfields Hospital from 1884 to 1898: from 1884 to 1888 there were 97 cases of sympathetic ophthalmia admitted out of a total of 10,676 in-patients, or 0·9 per cent.; from 1889 to 1893 there were 62 cases out of 10,095, or 0·61 per cent.; from 1894 to 1898 there were 47 cases out of 10,366, or 0·43 per cent. That little value can be attached to such

statistics is shown by the fact that the percentage comes out about the same whether all eye patients are reckoned or only in-patients, for practically all cases of sympathetic ophthalmia are treated as in-patients. It would be more advantageous to discover how often the disease occurs after perforating wound, but this is impossible since so many eyes are excised on account of their liability to induce the complaint. Ohlemann records 3 cases in 157 infected perforating wounds, of which 83 eyes were treated by neurectomy, excised or exenterated, thus making the percentage (2 per cent.) too low. Hobby reports 35 cases in 300 perforating wounds, and Knies computes 3 per cent., but the basis on which these computations are made is not evident.

As regards *sex* far more men than women are affected, which is of course referable to their greater exposure to the type of injury likely to produce the disease. As regards *age* children are undoubtedly more prone than adults, though Schirmer attributes this to the greater frequency of, perforating injuries amongst children. Römer found 64 patients under ten years of age amongst 264 cases of perforating wounds. My own impression is that this explanation is not completely satisfactory, but that children are really predisposed.

A *seasonal variation* in the incidence of the disease has been predicated. Brailey found 39 cases in summer to 13 in winter at Moorfields, Weber 13 in spring or summer to 6 in winter.

MOOREN.—Fünf Lustren ophth. Wirksankeit, Wiesbaden, 1882. O. BECKER.—Die Universitäts-Augenklinik in Heidelberg, Wiesbaden, 1888. OHLEMANN.—A. f. A., xxii, 1891. HOBBY.—Ophth. Rev., vi, 1887. RÖMER.—Z. f. prakt. Aerzte, 1898. BRAILEY.— Ophth. Congress, London, 1886. WEBER.—Dissertation, Zürich, 1895. *SCHIRMER.—In G.-S., vi, 2, 1900 (Bibliography).

The affection of the exciting eye is in the vast majority of cases a *perforating wound*. In nearly all cases some complication interferes with the rapid and reactionless healing of the wound, such as incarceration of the iris, lens capsule or ciliary body, the presence of a foreign body within the eye, etc. If suppuration occurs, especially if it goes on to panophthalmitis, the danger of sympathetic ophthalmia arising is slight. Usually there is plastic iridocyclitis, and the appearance of precipitates on the back of the cornea ("k. p.") is rightly regarded as of the gravest import.

As is well known wounds of the ciliary region—the so-called " dangerous zone "—have been looked upon as peculiarly susceptible. This is to be attributed merely to the effects of incarceration and the readiness with which traumatic cyclitis is set up, leading to delayed cicatrisation of the wound. Aseptic wounds of the ciliary body do not appear to be more prone to cause sympathetic ophthalmia than those in other parts of the eye. The same remarks apply to intra-ocular foreign bodies; if they are aseptic in the strictest sense of the word they are not likely to set up the disease. Occasional cases of purulent cyclitis followed by sympathetic are reported. Gunn found 4 in 47 cases of sympathetic ophthalmia at Moorfields; Schirmer 2 in 21 at Königsberg. The explanation on the bacterial theory of sympa-

thetic ophthalmia is that the pyogenic organisms prevent the multiplication of the specific organism in most cases in which it happens also to be present. Gifford's hypothesis that transmission is impeded by obstruction of the lymph channels with coagula is less likely.

Sympathetic ophthalmia is fortunately rare after operation wounds, and this is the case to a striking extent since the introduction of anti- and a-septic methods. Almost invariably when it occurs the operated eye has undergone such inflammatory changes as to cause blindness (*cf.* Mooren, v. Graefe, and Pagenstecher after reclination). Most attention has naturally been directed to cataract extraction, but the statistics vary within wide limits. Steffan records 28 eyes lost after cataract extraction in 300 cases, 6 developing sympathetic, *i. e.* 2 per cent. of the operated cases ; Eversbusch and Pemerl 154 lost eyes in 1420 extractions with 2 sympathetic, or 0·14 per cent. of the operated cases ; Bäuerlein 44 lost eyes in 860 extractions with no cases of sympathetic ophthalmia. There are at least 100 cases of sympathetic after extraction on record, though this is clearly no indication of relative frequency. In these cases posterior synechiæ, incarceration of iris in the wound, etc., are almost always mentioned. Anterior synechia of lens capsule is probably often present, though seldom recorded. Suppuration is rare, only slight inflammation still rarer (Webster, Galezowski, Milles, Becker). Some cases in which suppuration occurred at a late stage are due to " late infection," usually along the track of an anterior synechia or incarcerated iris or lens capsule (Leber, Wagenmann).

Sympathetic ophthalmia following a *perforated corneal ulcer* is rare, probably for the same reason that it is rare after perforating wound with suppuration. Gunn records a case.

*Discussion on Sympathetic Ophthalmitis, T. O. S., vi, 1886. Schweigger.— A. f. A., xv, 1885. Gepner.—C. f. A., ix, 1886. Alt.—Am. Jl. of O., 1884. Brailey.— Internat. Med. Congress, Berlin, 1890. Gunn.—R. L. O. H. Rep., xi, 1886. Milles.— R. L. O. H. Rep., x, 1882; xi, 1887. Gifford.—A. f. A., xvi, 1886. Schirmer.—A. f. O., xxxviii, 4, 1892; in G.-S., vi, 2, 1900. Steffan.—A. f. O., xxix, 2, 1883. Eversbusch and Pemerl.—A. f. A., xiii, 1884. Bäuerlein.—Ueber Staar u. Staaroperation, Wiesbaden, 1884. Webster.—T. Am. O. S., 1880. Galezowski.—Rec. d'O., 1880. Becker.—In G.-S., v, 1877. Wagenmann.—A. f. O., xxxv, 4, 1889.

A considerable number of cases of sympathetic ophthalmia have been described following diseases or injuries of one eye *without perforation of the globe*. All are open to much doubt and have been the subject of a vast amount of discussion. *Glaucoma* in itself is not a cause of sympathetic ophthalmia, and the same may be said of *herpes ophthalmicus* and *symblepharon*. More open to argument are the cases of intra-ocular tumour. This sequel has been reported in 30 cases of *sarcoma of the choroid* and 2 cases of glioma of the retina (Schirmer). The two latter (Steinheim, Walzberg) may be eliminated, since the description shows that they were pseudogliomata. Analysis of the cases of sarcoma of the choroid reduces the number of possible cases giving rise to sympathetic to very few, but these form an important and interesting group. In 7 of the 30 cases sympathetic irritation only was set up (Norris, Rémy, Salvioli, Noyes, Rosmini, Ovio).

Among the improbable cases are those of Berger, Rossander, and Amick, Pagenstecher, Knies, and Hotz.

Of the remaining cases, in five there was perforation of the globe, either by extra-bulbar extension of the tumour or by operative interference (Lawrence, Knapp, Steffan, Berlin and Schüppel, Leber and Krahnstöver, Pawel). In one case reported by Milles perforation took place only four days before the onset of inflammation in the other eye. Absence of perforation is definitely stated in the cases of Pagenstecher, Angelucci, Hirschberg, Lawford (extra-bulbar extension around the optic nerve), Deutschmann, and Nieden. There remain therefore 13 cases in which there was inflammation resembling sympathetic in the other eye, but in every one besides the tumour there was plastic inflammation of the uveal tract in the exciting eye. Most of the cases belonged to the rare group in which a sarcoma of the choroid is associated with shrinking of the eyeball. I have already dwelt at some length upon these cases (Vol. II, p. 524), which have been fully discussed by Leber and Krahnstöver, and I have also recorded a series of cases which I believe to have belonged to the initial stage of the group (Vol. II, p. 525). I am in complete agreement with the view of Leber and Krahnstöver that there is not sufficient evidence to show that sympathetic ophthalmia is ever set up by sarcoma of the choroid, though it may follow perforation of the globe through operative interference. That it ever follows perforation by extra-bulbar extension of the growth is highly problematical. Nieden's case in which cocci are said to have been demonstrated in the immediate vicinity of the tumour, there being no perforation of the globe, is too doubtful to authorise a diagnosis of endogenous infection. Such a conclusion must of course be admitted if further research confirms the presence of organisms in similar cases, but it by no means follows that the uveal inflammation thereby induced is truly sympathetic in nature.

Sympathetic ophthalmia has been attributed to *intra-ocular cysticercus* in some cases (Jakobson). In a case recorded by Pincus cataract extraction had been performed. There is no evidence that intra-ocular cysticercus alone is capable of inducing the disease.

Sympathetic ophthalmia following *subconjunctival rupture of the globe* has been recorded in 27 cases (Schirmer). In 3 there was sympathetic irritation only (Mooren, Guaita), in 3 the inflammation was not sympathetic (Robertson, Bresgen, Treitel), 2 are doubtful (Jacob, Bronner), in 8 the conjunctiva was not intact (Pagenstecher, Schrag, Ayres, Manolescu, Schmidt, Brudenell Carter, Kondos, Arlt). There remain 11 cases, in 9 of which there was extensive subacute or chronic uveitis, proved anatomically in 7 (Alt, Gunn, Sachs, Deutschmann, Müller, Meyer), doubtful in 2 (Barrier, Knapp). In 2 cases uveitis was absent clinically (Schröter, Schmidt). An additional case—subconjunctival dislocation of the lens—is recorded by Treacher Collins. Schirmer thinks that the uveitis is the essential factor in the production of sympathetic ophthalmia in such cases. Considering the extreme frequency of incarceration of the iris or ciliary body or both in subconjunctival rupture of the globe, I do not support this view. The presence of a minute wound of the conjunctiva or of an abrasion

through which exogenous infection might take place most probably explains the few doubtful cases in which true sympathetic ophthalmia may have occurred. Otherwise the injury is probably innocuous from this point of view.

Gonococcic infection (Hirschberg, Pflüger, Meyhöfer, Bunge, Knies, Wild, Deutschmann, Schirmer), tubercle (Manfredi, Brehmer), leprosy (Berger, Damsch), and so-called spontaneous inflammation in one eye cannot be accepted as causes of sympathetic inflammation in the other.

The *anophthalmic orbit* is not a source of sympathetic ophthalmia if those cases are eliminated in which the disease sets in within five weeks of excision of a dangerous eye. The Committee of the Ophthalmological Society of the United Kingdom collected 30 cases; Shaw and Stephenson have recently published others. In some of the recorded cases remnants of the eye were left in the orbit (Mooren, Lawson, Alt, Abadie). Schirmer found five cases in which iritis is attributed to wearing an artificial eye, but in one the exciting eye was removed for spontaneous iritis (Mooren); in two others there was an interval of seventeen and twenty-two years (Ferdinands), and in the others the diagnosis was almost certainly wrong.

Exenteration of the exciting eye is not an absolute guarantee against sympathetic ophthalmia, as in the cases of Dianoux, Tornatola, Waldispühl, Forget, Hotz, Abelsdorf, Pflüger, Cross. In all but one, however, sympathetic set in within three weeks of exenteration.

Sympathetic inflammation has also followed optico-ciliary neurotomy, as in the cases of Leber and Scheffels: in the former case the ends of the optic nerve were still separated, in the latter the eye was still anæsthetic, no regeneration of the ciliary nerves having yet taken place.

Sympathetic ophthalmia has also followed Mules's operation (Cross, and others).

Steinheim.—C. f. A., i, 1877. Walzberg.—K. M. f. A., xv, 1877. Norris.—Philadelphia Med. Times, 1874. Salvioli.—Ann. di Ott., iv, 1875. Noyes.—A. f. A., ix, 1880. Rosmini.—Ann. di Ott., xii, 1883. Ovio.—Ann. di Ott., xviii, 1889. Berger.—Beiträge zur Anat. des Auges, Wiesbaden, 1887. Pagenstecher.—Klin. Beobachtungen, Wiesbaden, 1862. Knies.—A. f. A., vi, 1877. Hotz.—Jl. Am. Med. Assoc., 1890. Lawrence.—Ophth. Rev., ii, 1866. Knapp.—Die intra-ocularen Geschwulste, Karlsruhe, 1868. Berlin.—A. f. O., xiv, 2, 1868. Schüppel.—Arch. f. Heilkunde, ix, 1868. *Leber and Krahnstöver.—A. f. O., xlv, 1, 1898. Pawel.—A. f. O., xlix, 1, 1899. Milles.—R. L. O. H. Rep., xi, 1887. Angelucci.—K. M. f. A., xvi, 1878. Lawford.—R. L. O. H. Rep., xi, 1887. Deutschmann.—Ueber die Ophthalmia migratoria, Hamburg, 1889. Nieden.—A. f. A., xxix, 1895. Jakobson.—A. f. O., xi, 2, 1865. Pincus.—A. f. O., xl, 4, 1894. Mooren.—Fünf Lustren ophth. Wirksamkeit, Wiesbaden, 1882. Guaita.—Ann. di Ott., vii, 1878. Argyll Robertson.—R. L. O. H. Rep., vii, 1871. Bresgen.—Wiener med. Woch., 1878. Treitel.—A. f. O., xxvi, 3, 1880. Jacob.—Brit. Med. Jl., 1870. Bronner.—T. O. S., xiv, 1894. Pagenstecher.—A. f. A., viii, 1879. Ayres.—A. f. A., vii, 1878. Manolescu.—A. d'O., v, 1885. Schmidt.—Dissertation, Giessen, 1895. Brudenell Carter.—In Nettleship, Ophth. Rev., i, 1880. Arlt.—Klin. Darstellung, 1881. Alt.—A. f. A., vi, 1877. Gunn.—R. L. O. H. Rep., xi, 1886. Sachs.—A. f. A., xx, 1889. L. Müller.—Ueber Ruptur der Korneoskleralkapsel, Leipzig, 1895. Barrier.—Ann. d'Oc., xxiv, 1850. Knapp.—T. Am. O. S., 1893. Treacher Collins.—R. L. O. H. Rep., xii, 1889. Schröter.—K. M. f. A., iv, 1866. Hirschberg.—Klin. Beobachtungen, Wien, 1874. Meyhöfer.—K. M. f. A., xv, 1877. Schirmer.—A. f. O., xxxviii, 4, 1892. Manfredi and Cofler.—A. d'O., i, 1881. Brehmer.—Dissertation, Königsberg, 1883. Damsch.—Virchow's Arch., xcii, 1883. Shaw.—T. O. S., xx, 1900. Stephenson.—T. O. S., xxi, 1901. Mooren.—Ophthalmiatrische Beobachtungen, Berlin, 1867; Ueber sympathische Gesichts-

störungen, Berlin, 1869. Lawson.—R. L. O. H. Rep., vi, 1868. Abadie.—A. d'O., iv, 1884. Ferdinands.—Brit. Med. Jl., 1898. Dianoux.—Soc. franç. d'O., 1886. Tornatola.—Ital. Ophth. Congress, Pisa, 1891. Waldispühl.—Dissertation, Basel, 1892. Forget.—A. d'O., xii, 1892. Hotz.—Am. Med. Assoc., 1893. Abelsdorf.—A. f. A., xxxiii, 1896. Pflüger. —Korrespondenzbl. f. Schweitzer Aerzte, 1896. Cross.—T. O. S., vii, 1887; xvii, 1897. *Schirmer.—In G.-S., vi, 2, 1900.

The *interval* between injury of the exciting eye and the development of sympathetic inflammation in the second eye varies from fourteen days to forty years. The accuracy of the diagnosis is greater in those cases in which the interval is short, *e. g.* within twelve weeks, than in those in which it is long. In the latter a fortuitous iridocyclitis may easily be mistaken for sympathetic ophthalmia if the other eye has been previously injured. Amongst 200 cases collected by the Ophthalmological Society of the United Kingdom (1886) 170 occurred after four weeks and before the end of the first year, 18 within four weeks, and 12 later than a year. The most dangerous period is from six to twelve weeks (Gunn—19 out of 28 cases). Schirmer states that the shortest interval in well authenticated cases is fourteen days (Nettleship, Schneider, Arlt, Gunn, Webster Fox, Ayres). Signs of sympathetic irritation occur earlier (Mooren, Alt, Vignaux, O. Becker, Nettleship, Cabannès and Ulry), but afford no proof of true sympathetic disease. The maximum interval it is really impossible to decide, for the longer the delay the less probable is the diagnosis. Amongst the most likely cases are those reported by Schirmer (15 years), Gunn (25 years), Vignaux (28 years), Sulzer (37 years), Weeks (42 years), Knapp (45 years). Weeks's case is doubtful; there had been a perforated ulcer in the first eye. In nearly all cases of long interval the first eye, which is usually shrunken, has become irritable and inflamed shortly before the onset of the sympathetic affection. This may be spontaneous or due to trauma, often scarcely appreciable. It is characterised by ciliary injection and tenderness to pressure. Anatomical examination of the exciting eye invariably (Brailey, Schirmer) shows signs of fresh inflammatory deposits, usually in the ciliary body or choroid, frequently in the iris, less often in cyclitic new formations. Too much stress has been laid upon the presence of bone in the choroid of the shrunken exciting globe. This is the inevitable sequel of phthisis bulbi; it is a purely degenerative phenomenon, and in a large number of cases is unassociated with pathological manifestations in the other eye. Moreover, shrunken eyes with bony choroids are often free from active inflammatory signs, both clinically and anatomically (Schirmer, Berger, Pooley, Parsons).

*Report of Committee on Sympathetic Ophthalmia, T. O. S., vi, 1886. Nettleship.—T. O. S., vi, 1886. Gunn.—R. L. O. H. Rep., xi, 1886. Schneider.—Dissertation, Würzburg, 1879. Arlt.—Klin. Darstellung, 1881. Webster Fox.—T. Am. O. S., 1885. Ayres.—A. f. A., xi, 1882. Mooren.—Ophthalmiatrische Beobachtungen, Berlin, 1867; Fünf Lustren ophth. Wirksamkeit, Wiesbaden, 1882. Alt.—A. f. A., vi, 1877. Vignaux.— De l'Ophthalmie sympathique, Paris, 1877. O. Becker.—Arch. f. Psychiatrie, xii, 1882. Cabannès and Ulry.—Clinique ophth., 1897. Sulzer.—Ann. d'Oc., cxxxviii, 1007. Weeks.—New York Eye and Ear Infirmary Rep., ii, 1894. Knapp.—A. f. A., ii, 1871. Brailey.—Internat. Med. Congress, Berlin, 1890. Berger.—Beiträge zur Anat. des Auges, Wiesbaden, 1887. Pooley.—Am. Jl. of O., 1884. *Schirmer.—In. G.-S., vi, 2, 1900. Rockliffe.—T. O. S., xxvii, 1907.

The *clinical manifestations of sympathetic ophthalmia* vary greatly, but almost always commence in the uveal tract, generally the ciliary body and iris. In the worst cases a severe plastic irido-cyclitis is set up, in the milder subacute or chronic " serous " cyclitis. It is impossible, and, indeed, misleading to draw a sharp line of distinction between the two forms as is often done. They are essentially different degrees of the same process, but it should be remembered that the more fibrinous the exudates poured out the more serious is the prognosis. Unfortunately the plastic form is the commoner. In many cases there is absolutely no prodromal sign (Hirschberg, Steinheim, Critchett, Tornatola, Schmidt-Rimpler, Schirmer), though some authors have stated that sympathetic irritation always precedes the attack (Ovio, Rolland). The first symptom is usually slight ciliary injection, but this may be overlooked and failure of vision may draw attention to the disease. By far the most important sign is the appearance of precipitates (" k. p.") on the back of the cornea. In every case of injury or intra-ocular operation the corneæ of both eyes should be most carefully examined for precipitates. If the injured eye does not quiet down rapidly and precipitates appear it is an extremely dangerous eye and very likely to set up sympathetic ophthalmia. The appearance of precipitates on the back of the cornea of the second eye is proof that sympathetic inflammation has already supervened. The other signs and symptoms are those of iridocyclitis in general, and need not be enumerated here. If it is possible to examine the fundus ophthalmoscopically slight papillitis, with blurring of the edges of the disc, may be seen in some cases. In the worst cases annular synechia is formed or total synechia, with retraction of the periphery of the iris. Occlusio pupillæ, complicated cataract, and phthisis bulbi lead to complete loss of the eye.

The milder "serous" iridocyclitis is comparatively rare in sympathetic ophthalmia : 4 times in 215 cases (Mooren, 1 in 116 ; Laqueur, 1 in 30; Gunn, 1 in 47 ; Schirmer 1 in 22). The prognosis is better in these cases, but plastic exudates may occur at any moment and the disease pass into the worse form. In these cases it may be possible to demonstrate choroidal inflammation, and there is no doubt that in most cases of sympathetic ophthalmia the disease is a uveitis affecting all portions of the uveal tract (v. Graefe, Jakobi, Steinheim, Dolschenko, Schmidt-Rimpler, Hirschberg, Caspar, Schirmer, Haab, etc.). Cases in which pronounced choroiditis was present are recorded by Colsmann, Rothmund and Eversbuch, Dolschenko, Leplat, Schirmer.

The frequency of papillitis in the ordinary uveal type of sympathetic ophthalmia is difficult to estimate. When present it is usually simultaneous in onset, though it may precede or follow the clinical manifestations of the uveal disease. A review of the cases tends to the conclusion that it is a sequel of uveal mischief, a view strongly supported by Becker's case in which there was clinically slight papillitis, anatomically severe choroiditis. Uncomplicated sympathetic papillo-retinitis is rare (Hirschberg, Pflüger, Vignaux, Harlan, Spalding, Eversbusch and Pemerl, Pooley, Brailey, Caudron, Gepner, Galezowski, Ayres and Alt, Hotz, Bjerrum, Mulder, Köhler). In 14

cases the interval of onset was less than two months in 8, fourteen weeks in 1, several years in 5 ; in the last group the exciting eye had recently become inflamed.

It is doubtful if primary sympathetic optic atrophy occurs. Schirmer could find only five uncomplicated cases, *i. e.* without uveitis, in the literature (Mooren, Krenchel, Roosa, Yvert, Rosenmeyer).

Sympathetic cataract does not occur ; cataract in sympathetic ophthalmia is a complication of the uveitis (*cf.* Krückow, Brière). The same remarks apply to so-called sympathetic detachment of the retina.

Sympathetic conjunctivitis has been described in five cases (Warlo-mont, Galezowski, Webster, Brailey) ; this is a mistaken diagnosis. Sympathetic keratitis is also unknown (Deutschmann, Schirmer).

There are seldom other manifestations of disorder in other parts of the body. Five cases are reported in which the eyelashes became white (Schenkel, Jakobi, Nettleship, Bock, Waren Tay), but there are other cases not recorded (*cf.* Vogt). It is difficult to explain this curious phenomenon. Severe headache (Haab), high temperature (Pflüger), delirium (Snellen, Deutschmann), meningitis (Story), etc., have been recorded.

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The *pathological anatomy* of sympathetic ophthalmia has already received some attention in the histological portion of this work (*v.* Vol. I, pp. 292, 353 ; Vol. II, p. 460). In order that the facts may be viewed in proper perspective from the pathogenic standpoint it is advisable to summarise them here, giving special regard to any distinctive characteristics.

As far as the limited number of observations on the sympathising eye permit, it may be stated that the changes are fundamentally the same in both eyes. Those in the exciting eye have been thoroughly

investigated, but it is necessary to eliminate the changes due to the injury or other cause of the disease as much as possible.
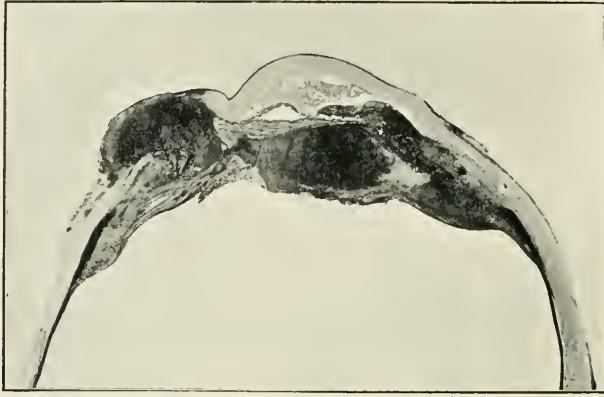
FIG. 816.—SYMPATHETIC OPHTHALMIA.
From a photograph by Coats.

In the exciting eye the iris usually shows a more uniform infiltration than other parts of the uveal tract, *i. e.* the more characteristic features of sympathetic inflammation are wanting. In some cases it may be

FIG. 817.—SYMPATHETIC OPHTHALMIA.
From a photograph by Coats, showing nodular iritis (*cf.* Fig. 192).

free from the sympathetic infiltration, in others it is equally affected, and in a few it is most involved, though never alone (Fuchs). The

infiltration commences in the posterior layers, sometimes even behind the sphincter. First there are isolated nodules (*v.* Vol. I, p. 292), later these coalesce to a uniform lamina. Further extension is chiefly backwards, invading any retro-iridic exudate which may be present, the retinal layer being forced forwards and disintegrated. The anterior layers escape until a late stage. The cellular constituents are first lymphocytes, later epithelioid cells, which continue to increase and invade the anterior layer of retinal epithelium. The posterior layer is less vulnerable, but finally succumbs. Giant cells are less common in the iris than in other parts of the uveal tract. They lie between the epithelioid cells and appear first in the posterior layers, finally invading the whole thickness of the iris. In the early stages the blood-vessels appear as bright rings in the cellular mass, but they ultimately degenerate and disappear, being replaced by new formed endothelial channels. These changes differ from the uniform œdema, fibrinous exudation, and infiltration with lymphocytes and leucocytes of traumatic iridocyclitis.

The ciliary body is always involved, often most severely, rarely only slightly. The earliest signs are best seen in the posterior part of the plicated zone. The tissue beneath the retinal epithelium is denser than that outside, bordering the ciliary muscle. It is in the outer looser layer, internal to the muscle, that infiltration first appears, consisting of lymphocytes. In the inner denser zone epithelioid cells appear, which are also present in the outer zone, masked by the lymphocytes. The multiplication of epithelioid cells outstrips that of lymphocytes. The ciliary muscle generally escapes invasion for a long period. As in the iris extension is chiefly towards and into the retinal layer, the pigment granules escape, and become aggregated together, whilst the epithelial cells are broken up. Fuchs considers that not only the mesoblastic fixed cells but also the depigmented epithelial cells give rise to epithelioid cells. The non-pigmented retinal epithelium, like the posterior layer of iridic retinal epithelium, is more resistant than the outer layer. Giant cells are derived from the epithelioid cells and therefore appear first in the innermost layer of the stroma. The vessels undergo the same changes as in the iris, the veins succumbing first. There is the same difference·between this process and that in endophthalmitis affecting the iris. In the latter the innermost layers of the stroma suffer first, especially the tips of the ciliary processes, which are infiltrated with lymphocytes and polymorphonuclear leucocytes, whilst in the former the bays between the ciliary processes are first attacked.

The choroid is found anatomically to be generally the seat of most inflammatory reaction (Fuchs). In 35 cases it was intensely inflamed in 22, and in 12 more than the anterior segments of the uveal tract, which were scarcely involved at all in 3 (Fuchs): in the remainder of the 22 cases the infiltration of the choroid was certainly not less than that of the iris and ciliary body. The infiltration is greater in the posterior parts than near the ciliary body, except when both are very extensively involved: it is often absent near the ora serrata, isolated nodules appearing behind this, coalescing into a lamina still farther back.

Immediately around the disc the infiltration may again diminish. The first deposits occur in the layer of large vessels, especially along the
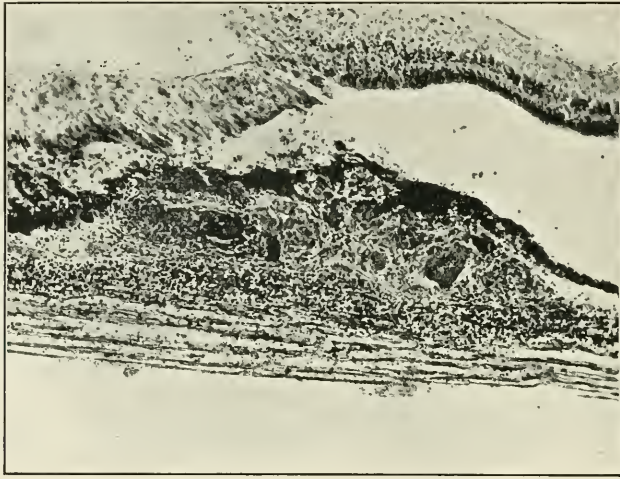
FIG. 818.—SYMPATHETIC OPHTHALMIA.
From a photograph by Coats, showing retina and choroid, with giant cells in the latter.

veins. Slight aggregates of lymphocytes are seen in the layer of medium sized vessels, but scarcely any changes in the chorio-capillaris. Even
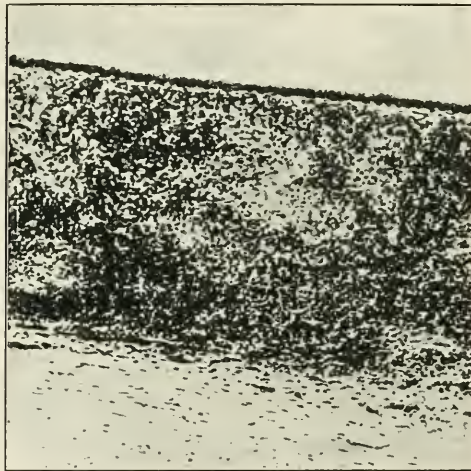
FIG. 819.—SYMPATHETIC OPHTHALMIA.
From a photograph by Coats, showing choroiditis.

in the later stages when the infiltration becomes diffuse the chorio-capillaris long escapes (Brailey, Fuchs). The early deposits are

nodules of lymphocytes, not, however, sharply delimited. The first epithelioid cells occur usually in Haller's layer, often occupying the centre of the lymphocytic nodules, which may be almost entirely replaced by them. In rare cases they are almost, if not quite, absent (Fuchs). They are always most numerous where the infiltration is densest. Giant cells follow the epithelioid cells, and as in other parts of the uveal tract are sometimes very numerous, sometimes—in half of Fuchs's cases—absent. With the advance of the process the choroidal stroma degenerates ; the choroid may even become quite depigmented, but even when the chromatophores have disappeared the epithelioid and giant cells may be pigmented, sometimes deeply. With the destruction of the stroma the membrane of Bruch also disappears. The lymphocytes pervade the walls of the veins, finally encroaching upon the lumen. The arteries are more resistant, but succumb eventually, though the details of the degenerative process vary in different vessels. It will be noticed that the distribution of the infiltration is quite different in ordinary endophthalmitis (v. Vol. II, p. 446), in which the anterior and posterior parts of the choroid usually suffer first and most severely.

The changes in other parts of the eye are always relatively slight, but they demand special consideration in order that no fact of importance from the point of view of pathogenesis may be overlooked. Dalén has drawn attention to nodules on the inner surface of the choroid, present only rarely, but sometimes in cases in which the choroidal affection was slight. They occurred in four of Fuchs's cases. A few of the pigment epithelial cells swell and proliferate, forming several layers, the outer of which lose their pigment. A few lymphocytes are present, said to be derived from the choroid even in the absence of any aperture in Bruch's membrane. The nodules may increase considerably in size, the cells becoming lengthened out into spindles. The overlying retina shows no trace of inflammatory reaction. In rare cases the cellular infiltration of the choroid extends inwards, encroaching upon the sub-retinal space. This is much less common than extension outwards into the supra-choroidal space, which is indeed the rule in advanced cases. The space is thus obliterated first in the posterior part. Lymphocytes form a mantle to the perforating ciliary vessels and nerves and venæ vorticosæ. Schirmer found the anterior, Fuchs the posterior ciliary vessels most affected, but still more so are the vorticose veins, along which epithelioid and giant cells often form large nodules. The intra-scleral nodules may coalesce and lead to rupture of the sclerotic, but this is apparently commoner in the sympathising eye (Schirmer, Ruge, Blaschek). Extra-ocular proliferation is more frequent.

Increase in the number of mast cells is a conspicuous feature, not only in the uveal tract but also in the sclerotic, episcleral tissue, and optic nerve (Schirmer, Uhr, Pincus, Meyer). Their intensely stained granules are very liable to be mistaken for cocci, especially when they are set free by disorganisation of the cells, as is often the case (Axenfeld, Uhr).

In the retina there is nearly always cellular infiltration around the

vessels, especially the veins, but typical sympathetic infiltration is quite rare, following rupture of the membrane of Bruch. Fuchs found it only in one case in which the retina was detached and embedded in a cyclitic mass. Other observers describe it more frequently (Ruge).

The changes in the optic disc are slight, consisting usually only of perivascular infiltration. The densest infiltration is generally immediately behind the lamina cribrosa, and diminishes farther back. Infiltration of the vaginal space is also usually limited to the anterior portion. Typical nodules are extremely rare but have been found by Schirmer and Ruge.

Panophthalmitis in the exciting eye has been described only by Schirmer in two cases, and he is of the opinion that it is due to mixed infection. The tendency in all cases is for organisation to take place. In the stroma of the uveal tract the cellular elements gradually disappear, the epithelioid and giant cells completely, and are replaced
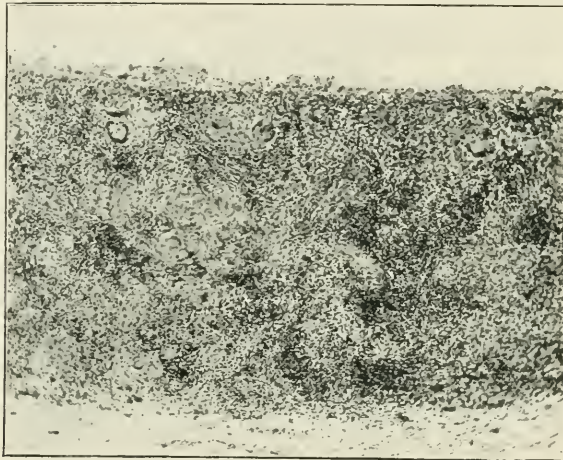
FIG. 820.—SYMPATHETIC OPHTHALMIA.
Showing choroiditis in sympathising eye, with giant cells.

by fibrous tissue, which eventually resembles the sclerotic in structure. This retrograde metamorphosis may be irregularly distributed, so that parts are being converted into scar tissue whilst other parts show active infiltration. The superficial layers of the uveal tract suffer least in sympathetic ophthalmia, thus differing from the condition in long-standing endophthalmitis. The earliest signs of retrogression were found by Fuchs in a case six weeks after the injury, and appeared first in the anterior layers of the iris.

Fuchs does not consider that plastic exudation is a characteristic feature of the sympathetic process *per se*. It may be absent even in severe cases, though it is almost always present in the exciting eye. It is probably due to the injury or other cause of sympathetic ophthalmia, as is shown by the following considerations. The plastic exudation nearly always shows by its anatomical features—dense fibrous tissue

free from nuclei—that it is of older date than the sympathetic inflammation. The endophthalmitis and the sympathetic inflammation progress independently side by side; there is almost an inverse ratio between them (Fuchs). The plastic deposits and the sympathetic infiltration are often most marked in different parts of the eye. The two forms of inflammation were first carefully dissociated by Straub. It is a striking confirmation of the essential independence and difference of the two processes that Fuchs was able to separate his 200 cases of eye excised for injury accurately into those which had set up sympathetic ophthalmia and those which had not from examination of the sections alone without the help of the histories.

The sympathising eye shows similar changes to those found in the exciting eye in the ten cases which are on record (W. J. Collins, Deutschmann, Zimmerman, Grunert—two cases, Asayama, Schirmer, Blaschek, Welt, Milles). In eight of these cases there was little or no plastic exudation, thus supporting Fuchs's view that this is an epiphenomenon. Much attention has been directed on theoretical grounds to the condition of the papilla and optic nerve in these eyes. In Collins's case there was considerable infiltration of the disc, anterior part of the nerve and vaginal space. The retina showed diffuse infiltration and dense mantles of lymphocytes around the vessels. Deutschmann states that the infiltration in the nerve is greatest in the pia, arachnoid, and intervaginal space, slight in the dural sheath. It must be borne in mind that his views on this subject are apt to be biased. In Zimmermann's case there was meningitis, and in one of Grunert's nephritis and a cerebral tumour, so that these cases are of no value in this connection. According to Deutschmann the chiasma is even more infiltrated than the nerve.

The blood shows no conspicuous changes, such as leucocytosis, in sympathetic ophthalmia (Terrien and Cantonnet).

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The *bacteriology* of sympathetic ophthalmia is negative as far as the discovery of a pathogenic organism is concerned. Ayres and Alt, Nordenson, Randolph, Haab, Schmidt-Rimpler, Pflüger, Schanz, Kuhnt, Trousseau, Poncet, Ohlemann, Uhthoff, Schirmer, Greeff, Bocchi, Bourgeois, Peppmüller, Treacher Collins, Bach, Runck, Axenfeld, Uhr, Shaw and others, have failed to find any organism in the exciting eye by the most varied methods and the use of all kinds of culture media. Pyogenic organisms have been found—staphylococci by Deutschmann, Waldispühl, Limbourg and Levy, streptococci by

Angelucci, Limbourg and Levy. Sattler and Stilling found cocci, differing only slightly from staphylococci in sympathising eyes. It is highly improbable that ordinary pyogenic organisms should be the cause; it is not surprising that they should be found in the injured eye, but their presence in the sympathising eye requires ample confirmation before it can be accepted as an established fact. Deutschmann alone found staphylococci in almost all his cases, both microscopically and by culture—in 9 exciting and 5 sympathising eyes. Organisms, generally cocci, sometimes bacilli, have been more frequently described in microscopical sections (Leber, Snellen, Abraham and Story, Berger, Basevi, Berry, Wagenmann, Secondi, Pincus, Angelucci, Meyer, Zimmermann). No importance is to be attached to the presence of so-called cocci unless the author has carefully considered the possibility of having mistaken them for the granules of mast cells (v. p. 1243). Even then the observation must be accepted with great reserve. It is noteworthy that the organisms have often been found in parts of the eye least affected by the specific inflammation, i. e. not in the uveal tract, but in the scar episcleral tissue, optic nerve, etc. (Deutschmann, Pincus, Meyer) or vitreous (Abraham and Story). Special attention has been directed to the presence of organisms in the optic nerves and chiasma as evidence bearing upon the migratory theory (vide infra). Deutschmann's observations will not bear exhaustive analysis, and in Zimmermann's case there was purulent meningitis.

Implantation of portions of affected tissues in the eyes of animals have generally given negative results (Greeff, Schirmer). Schirmer has obtained positive results with portions of ciliary body from an exciting and from a sympathising eye in the anterior chamber of a rabbit. In both cases a chronic uveitis was set up leading to commencing phthisis bulbi in four to five weeks, when the eyes were enucleated. The rabbit's other eye remained normal. Schirmer regards this experiment as conclusive evidence of the presence of organisms in the sympathising eye.

Raehlmann's observation of bacilli and minute motile particles by the ultramicroscope cannot be regarded as conclusive; bodies of the dimensions described could be seen by an oil-immersion objective.

zur Nedden found that blood from a patient with sympathetic ophthalmia, when injected into a rabbit's vitreous, set up more inflammation than normal blood. The vitreous from the inoculated animal set up inflammation in a second animal, and in the vitreous of the fourth animal from such a series he found a pseudo-diphtheria bacillus. The organism was cultivated with difficulty, but culture injected into the carotid often set up a chronic metastatic inflammation in the eyes. Brons in Axenfeld's laboratory was unable to confirm zur Nedden's results.

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The **theories as to the pathogenesis** of sympathetic ophthalmia are classified by Schirmer as follows: (1) Pure nerve theories: (*a*) optic nerve theory; (*b*) ciliary nerve theory. (2) Pure bacterial theories: (*a*) transmission by metastasis; (*b*) transmission by reversed venous flow; (*c*) transmission by lymph channels. (3) Combined theories: (*a*) Meyer's theory; (*b*) Schmidt-Rimpler's theory. (4) Toxic theory.

The *optic nerve theory* is based upon the striking anatomical fact that the eyes are connected by the optic nerves through the chiasma. It is difficult from the writings of le Dran, Himly, Mackenzie and others, who lay stress upon this connection, to be certain that they supported a purely nervous agency in the transmission. Mackenzie says: " It is extremely probable that the retina of the injured eye is in a state of inflammation which is propagated along the corresponding optic nerve to the chiasma, and thence the irritation which gives rise to the inflammation is reflected to the retina of the opposite eye along its optic nerve." He, however, also discussed the possibilities of transmission by the blood or the ciliary nerves. H. Müller's objection that the optic nerve is often completely atrophic applies only to a purely nervous mode of transmission. Mooren's suggestion that the irritation travels by the trigeminal to the optic nerve and thence to the opposite trigeminal needs only to be mentioned.

The *ciliary nerve theory* states that irritation of the ciliary nerves is transmitted to the opposite ciliary nerves, the impulses setting up changes in the other eye which eventually amount to inflammation. The exciting irritation may be mechanical, chemical (Bocchi), or bacterial. The theory was originated by H. Müller. Many observers have found evidence of inflammatory changes in the ciliary nerves (Schmidt-Rimpler, Goldzieher, Krause, Uhthoff, Berger, and others), whilst in some cases it has been absent (Berger, Brailey, Schirmer, and others). It would be surprising if the ciliary nerves escaped either in the exciting or the sympathising eye when the disease is fully developed, and the evidence is of no importance in deciding the merits of the theory. Proof of primary incidence of inflammation in the ciliary nerves of the sympathising eye would of course be confirmatory, but it is wanting. Experimental evidence is contradictory. Mooren and Rumpf irritated the exposed iris of a rabbit with spirit of mustard and the Paquelin cautery; the opposite iris showed anæmia; with ether the opposite iris showed hyperæmia. Jesner cauterised the corneal margin with the silver stick; the aqueous of the other eye showed fibrinous coagula. Wessely repeated Jesner's experiments, estimating the albuminous content of the aqueous of the other eye; in 32 experiments there was no abnormal increase. Bach, under similar conditions, found minute coagula and extravasations of blood in the anterior and

posterior chambers and in the periphery of the vitreous; he, however, denies his adherence to the ciliary nerve theory. Shaw kept up jequirity conjunctivitis and mechanical injury for six months; slight infiltration of the uveal tract of the other eye occurred, but was not progressive. These experiments are crude; even Wessely's are inconclusive for any increase in albumin would be within the limits of experimental error. This objection does not hold for Römer's very interesting and delicate investigations of the transudation of hæmolysins in immunised animals under similar conditions (v. Vol. III, p. 1036). He has definitely disproved the ciliary nerve theory in so far as ordinary irritants are concerned. It does not absolutely follow that some specific irritant, present in sympathetic ophthalmia only, might cause irritation in the other eye by way of the ciliary nerves, but it is in the highest degree improbable.

The *theory of bacterial transmission by metastasis* was mentioned by Mackenzie, but the credit of advancing it definitely rests with Berlin. As it appears to me to be the most probable theory it will be discussed in detail after the other theories have been reviewed.

The *theory of bacterial transmission by reverse venous flow* was advanced by Arnold, who suggested that bacteria might pass backwards into the cavernous sinus and then be carried to the opposite eye by the veins owing to reverse current induced by coughing, etc. Obvious objections to the theory are the absence of evidence of the importance of any such causes of reversed flow, the difficulty of bacteria being forced into the small ciliary tributaries, the extreme rarity of bilateral sarcoma of the choroid such as might be expected on the same grounds, etc.

The *theory of bacterial transmission by the lymph channels* was originated by Leber and strongly supported by Deutschmann under the designation "ophthalmia migratoria" for the disease. The most obvious lymph channel is the intervaginal space of the optic nerve, which communicates indirectly with that of the opposite nerve by way of the chiasma. Of subcutaneous lymph channels over the root of the nose (Scheffels) we know nothing. The chief support of the theory, apart from the extreme probability of a bacterial origin and the existence of a definite anatomical communication between the two eyes, is derived from questionable experimental evidence. It must be clearly borne in mind that this evidence deals entirely with ordinary pyogenic organisms, which, as has been seen, scarcely ever set up sympathetic ophthalmia, and have scarcely ever been observed in the sympathising eye. I am in substantial agreement with Römer when he says that "the question whether any conclusion can be drawn as to the pathogenesis of sympathetic ophthalmia in man from experiments on animals with any pyogenic organism must be answered in the negative." Deutschmann (1882) first obtained papillitis in an eye by injecting spores of aspergillus fumigatus into the vitreous of the other eye; inflammatory changes were found in the optic nerves and chiasma. He obtained similar results with croton oil, and concluded that the disease was caused by the transmission of bacterial metabolic products. With staphylococci he obtained papillitis in twelve of

thirty-four cases, and in two of thirty-five of a later series ; there was never uveitis in the " sympathising " eye. Staphylococci could be traced in the intervaginal space, about the chiasma, and in the opposite intervaginal space, and in many cases the animals died of general septicæmia. Gifford repeated Deutschmann's experiments, but never saw any changes in the other eye ; he obtained them in three of twenty-five cases with anthrax bacilli. Mazza obtained negative results with staphylococci, and found them in the sheath only when the animals died of meningitis. Sattler obtained negative results with a staphylococcus derived from a human sympathising eye. Negative results were obtained by Randolph, Ulrich, Limbourg and Levy, Schirmer, Angelucci, and others. Greeff found cocci in the opposite nerve only when they were also present in the blood. Alt confirmed Deutschmann's experiments with croton oil. Basevi had positive results with bacilli cultivated from an exciting eye, negative with cocci. Parisotti found papillitis in eight out of thirty experiments with staphylococci injected subcutaneously ; three of these died of meningitis. E. Meyer obtained iridocyclitis, but found that the other rabbits in the same hutch were similarly affected. Moll found with *Bacillus pyocyaneus* that the organisms could often be recovered from the aqueous, but this occurred whether optico-ciliary neurotomy was performed or not. Bellarminoff and Selenkowsky obtained negative results with staphylococci, *B. coli*, *B. prodigiosus*, and *B. pyocyaneus*. It will be realised, therefore, that the negative results far exceed the positive, and that the latter are of little if any value as evidence in favour of the theory.

Very little evidence can be brought forward in favour of the migratory theory from clinical observations. In Pagenstecher's case the patient died of meningitis three days after enucleation. The cases of Deutschmann, Zimmermann, Schirmer, and Grunert, in which post-mortem examinations were made, are in no way conclusive.

The clinical evidence against the theory is strong. Apart from the infrequency of sympathetic disease after septic wounds and the difficulty of the long latent period in some cases it has been seen that the disease may follow early optico-ciliary neurotomy. There is extremely rarely any sign of meningitis, such as might be expected. Sympathetic papillitis is very rare, whilst it is the only experimental evidence which can be adduced in favour of the theory.

Meyer's theory is that ciliary nerve irritation only causes sympathetic ophthalmia if the second eye already contains pathogenic organisms : if the eye is normal sympathetic irritation is set up.

Schmidt-Rimpler's theory is that ciliary nerve irritation prepares the way for the unknown infective agent, which is innocuous unless the circulation and nutrition of the second eye have been thus impaired. It is clear that the theory stands or falls with the proof or disproof of any such reflex influence upon the circulation and nutrition of the other eye. Admitting this reflex influence for the sake of argument, there is no analogy for any such reflex predisposition to bacterial invasion or toxic affection. It has been seen that the experimental evidence is against the reflex ciliary nerve theory. Other experiments have been

made from the point of view of Schmidt-Rimpler's theory. Moll injected cultures of pyocyaneus bacilli intra-venously in rabbits and cauterised the cornea with the silver stick or introduced sterile copper into the anterior chamber. In 77·3 per cent. the bacillus was found in the aqueous of both eyes. When one eye was not irritated the aqueous contained the bacillus in 23·1 per cent. Such investigations, in which virulent septicæmia is produced, are of little value as regards sympathetic ophthalmia. Panas found that a rabbit's eye succumbed to bacterium coli injected subcutaneously if nicotin was injected into it.

The *toxic theory* is based chiefly upon the fact that micro-organisms have very rarely been found ; it has, therefore, been concluded that the inflammation is caused by their metabolic products (Gorecki, Rosenmeyer, Bocchi, Praun and others). The strongest clinical argument against the theory is that the disease continues after removal of the exciting eye, and may even commence under these circumstances. The view that ordinary sympathetic uveitis is bacterial whilst sympathetic papillitis is toxic in origin seems to me to be highly improbable, though it is accepted by Schirmer.

Of the many theories which have been briefly reviewed the most probable seems to me to be the bacterial metastatic theory. Many of the objections raised against it by Leber and other observers have lost much of their force with the advance of bacteriology. The view that the conditions of development of organisms in the eye are the same as elsewhere in the body (Leber) is no longer tenable as a general law. Different tissues and organs of the body undoubtedly show specific characteristics which make them suitable for special organisms, as is exemplified for streptococci, tetanus bacilli, typhoid bacilli, etc. Most of the facts point to sympathetic ophthalmia being a disease of bacterial origin, and if the virulence of the organisms and the varying conditions of resistance of the tissues are taken into account the variations in latent period and many other difficulties are abolished. It is probable that the organism is pathogenic only for the eye and is innocuous to other parts of the body, though there the conditions are not so adverse as to prevent its propagation. zur Nedden has advanced reasons to show that the organisms are ultravisible by present methods, but that they are too large to pass through a Berkefeld filter. It is characteristic of organisms which set up metastatic inflammation, *e. g.* tubercle, leprosy, etc., that the disease is sub-acute or chronic, not purulent. Even virulent organisms may be inactive when circulating in the blood-stream. It is a striking fact that saprophytes can set up serious inflammation in the eye, whilst they are innocuous in other parts of the body. It is not to be concluded that the specific organism of sympathetic ophthalmia is an ordinary saprophyte, but that it is pathogenic for the eye whilst indifferent to the rest of the body, and that it reaches the eye by way of the blood.

Bacteriological investigation has shown that in almost all infectious diseases some of the organisms escape into the circulation; otherwise the facts of immunity—the development of active immunity, the production of specific antibodies, especially bacteriolysins—would be incom-

prehensible (*v.* Vol. III, Chap. XIX). It is the presence of specific antibodies which often makes these circulating bacteria innocuous, *e. g.* the presence of anti-staphylotoxin in normal plasma prevents metastasis in a staphylococcic panophthalmitis. The cases of long latent period in the development of sympathetic ophthalmia have been adduced against the metastatic theory. That organisms can remain viable in the circulation for a long period is proved by many experiments. Wysso-kowitsch showed that living spores of saprophytes could be recovered from the liver and spleen seventy-eight days after injection. It would appear that the capillaries of these organs filter them off from the blood.

No other theory explains the uveitis which characterises most cases of sympathetic ophthalmia so well. The analogy of tubercle, syphilis, etc., is obvious, and the accumulation of evidence of the topical selec-tion of different bacteria (*v.* p. 1222) favours the theory. Sympathetic choroiditis (Hirschberg, Haab) can be explained with difficulty on any other hypothesis. The fact that sympathetic ophthalmia manifests itself as a uveitis both in the exciting and in the sympathising eye points to the same *causa causans.* The organism multiplies in the exciting eye ; if this is removed early infection does not occur, but if organisms have escaped in quantity into the circulation sympathetic ophthalmia may follow. The conditions of existence in the circulation and in other organs of the body are relatively bad, as is shown in the case of other organisms. It is probable that early encapsulation may occur in the exciting eye. As is well known bacteria may long lie dormant but viable in this condition. Slight injury or other cause may set them free, thus explaining the occurrence of sympathetic ophthalmia long after the first injury, though preceded at a short interval by inflamma-tion in the exciting eye.

LE DRAN.—Traité, etc., Amsterdam, 1741. HIMLY.—Die Krankheiten, etc., Berlin, 1843. MACKENZIE.—Treatise, London, 1830, 1835, 1839, 1854. H. MÜLLER.—A. f. O., ii, 1, 1858. MOOREN.—Ueber sympathische Gesichtsstörungen, Berlin, 1869. BOCCHI.—Internat. Congress, Rome, 1894. SCHMIDT-RIMPLER.—K. M. f. A., xii, 1874. GOLDZIEHER.—K. M. f. A., xv, 1877. KRAUSE.—A. f. A., x, 1881. UHTHOFF.—A. f. O., xxix, 3, 1883. BERGER.—Beiträge, Wiesbaden, 1887. BRAILEY.—T. O. S., v, 1885. SCHIRMER.—A. f. O., xxxviii, 4, 1892. MOOREN AND RUMPF.—C. f. med. Wissenschaft, 1880. JESSNER.—Pflüger's Archiv, xxiii, 1880. WESSELY.—A. f. O., l, 1900. BACH.—A. f. O., xlii, 1, 1896. SHAW.—Brit. Med. Jl., 1898. RÖMER.—A. f. O., lv, lvi, 1903. BERLIN.—Volkmann's Sammlung klin. Vorträge, 1880. ARNOLD.—A. f. path. Anat., cxxiv, 1891. LEBER.—A. f. O., xxvii, 1, 1881 ; lviii, 1904. SCHEFFELS.—K. M. f. A., xxvii, 1890. *RÖMER.—A. f. O., lv, 1903; A. f. A., liv, lv, lvi, 1906. GOLOWIN.—A. d'O., xxv, 1905. PUSEY.—A. f. A., lii, 1905. SANTUCCI.—Riv. Ital. di Ott., 1906. DEUTSCHMANN.—A. f. O., xxviii, 2, 1882; xxix, 4, 1883 ; xxx, 3 and 4, 1884; Ueber die Ophthalmia migratoria, Hamburg, 1889; B. z A., 1893; C. f. A., xxiii, 1899. GIFFORD.—Am. Jl. of O., 1887. MAZZA.—Ann. di Ott., xvi, 1887; Internat. Ophth. Congress, Heidelberg, 1888. SATTLER.—Internat. Ophth. Congress, Heidel-berg, 1888. RANDOLPH.—A. f. A., xxi, 1890. ULRICH.—B. d. o. G., 1891. LIMBOURG AND LEVY.—A. f. exp. Path. u. Pharm., xxviii, 1890 ANGELUCCI.—Arch. di Ott., iv, 1896. GREEFF.—A. f. A., xxvi, 1893. ALT.—Am. Jl. of O., 1884. BASEVI.—Ann. di Ott., xix, 1890. PARISOTTI.—Internat. Congress, Berlin, 1890. GAYET.—A. d'O., x, 1890. E. MEYER.—B. d. o. G., 1891. MOLL.—C. f. A., xxii, 1898. BELLARMINOFF AND SELENKOWSKY.—A. f. A., xliv, xlv, 1902. PAGENSTECHER.—K. M. f. A., xi, 1873. ZIMMERMANN.—A. f. O., xlii, 2, 1896. *SCHIRMER.—In G.-S., vi, 2, 1900. GRUNERT.—K. M f. A., xxxviii, 1900 ; xxxix, 1901. PANAS.—A. d'O., xvii, 1897. GORECKI.—Soc. franç. d'O., 1891. ROSEN-MEYER.—A. f. A., xxviii, 1893. PRAUN.—Die Verletzungen des Auges, Wiesbaden, 1899. ROGER.—Comptes rendus de la Soc. de Biol., 1889. WYSSOKOWITSCH.—Z. f. Hygiene, 1886. HIRSCHBERG.—C. f. A., xix, 1895. HAAB.—B. d. o. G., 1897. RAEHLMANN.—Deutsche med. Woch., 1904. ZUR NEDDEN.—A. f. O., lxii, 1906.

CHAPTER XXVII

SYMPTOMATIC DISEASES OF THE EYE

SYMPTOMATIC diseases of the eye include all those pathological changes in the eye which are induced by diseases which primarily attack other parts of the body. Many of the conditions already discussed should more properly be classified under this heading, but on account of their histological characters, the methods of experimental investigation applied to them, etc., they have been more conveniently treated elsewhere. It is beyond the scope of this treatise to enter fully into the pathology of all the diseases which may have ocular manifestations (*see* Schmidt-Rimpler, Groenouw, Uhthoff, and others). Only those conditions in which the ocular manifestations are pronounced or which have some special bearing upon ophthalmic pathology will be considered.

JACOBSON.—Beziehungen der Veränderungen u. Krankheiten des Sehorgans zu Allgemeinleiden u. Organerkrankungen, Leipzig, 1885. BERGER.—Les Maladies des Yeux dans leurs Rapports avec la Pathologie générale, Paris, 1892. KNIES.—Die Beziehungen des Sehorgans u. seiner Erkrankungen zu den übrigen Krankheiten des Körpers u. seiner Organe, Wiesbaden, 1893. *SCHMIDT-RIMPLER.—Die Erkrankungen des Auges in Zusammenhang mit anderen Krankheiten, Wien, 1905. *GROENOUW, UHTHOFF.—In G.-S., xi, 1904– . GOWERS.—The Ophthalmoscope in Medicine, London, 1904.

DISEASES OF THE RESPIRATORY TRACT.

Every respiration is accompanied by changes in the intra-vascular blood-pressure, the intra-ocular pressure (*v.* Vol. III, p. 1055), and the pupil. During inspiration there is slight dilatation, during expiration slight constriction of the pupil. In asphyxia the pupil is first constricted, then widely dilated and immobile, the changes being due chiefly to action of the asphyxial blood on the nerve centres. The effects of Cheyne-Stokes' respiration vary in different cases (Robertson, Gibson, Thiemich, and others).

Air may be forced into the nasal duct by strong expiration with closed mouth and nose, sneezing, etc. (v. Graefe, Rau, Starcke ,Malgat). Blowing the nose has caused dislocation of the eye (Dépontot, Knies), orbital emphysema (Schanz), orbital hæmorrhage (Berlin), etc.

ROBERTSON.—Lancet, 1886. GIBSON.—Cheyne-Stokes' Respiration, Edinburgh, 1892. THIEMICH.—Jahrbuch f. Kinderheilkunde, xlvii, 4, 1898. v. GRAEFE.—K. M. f. A., vi, 1868.

Rau.—A. f. O., i, 2, 1855. Starcke.—A. f. klin. Med., vii, 1870. Malgat.—Rec. d'O., 1890. Dépontot.—Jl. de Méd., 1885. Berlin.—K. M. f. A., xi, 1873.

In **whooping = cough** conjunctival hæmorrhages are common (Clement Lucas); rarer are orbital hæmorrhage with exophthalmos (Landesberg), hyphæma (Warner), retinal hæmorrhage (Landesberg), intra-ocular hæmorrhage and detachment of the retina (Teillais), subluxation of the lens (Landesberg). Partial or complete blindness may follow cerebral hæmorrhage or involvement of the optic nerve in the orbit. Homonymous hemianopia (Silex, Fritsche, Jacoby), amblyopia during the paroxysms (Steffen), iridocyclitis (Chronis), optic neuritis with subsequent atrophy (Alexander), paralysis of the sixth and seventh nerves (Craig), etc., have been recorded.

Clement Lucas.—Guy's Hosp. Rep., xix, 1874. Knapp.—A. f. A., v, 1876. Steffen.—Ziemssen's Handbuch, iii, 1876. Landesberg.—Med. and Surg. Reporter, xliii, 1880. Callan.—Am. Jl. of O., i, 1884. Alexander.—Deutsche med. Woch., 1888. Silex.—Berliner klin. Woch., 1888. Fritsche.—Jahrb. f. Kinderhk., xxix, 1889. Troitzky.—Jahrb. f. Kinderhk., xxxi, 1890. Jacoby.—New York Med. Jl., 1891. Warner.—Jl. of Ophth., Otol., and Laryng., v, 1893. Teillais.—Rec. d'O., 1895. Craig.—Brit. Med. Jl., 1896. Chronis.—K. M. f. A., xliii, 1905.

In **pneumonia** herpes corneæ is the commonest complication (*see* " Neuroparalytic Keratitis "). Metastatic ophthalmia may occur (Herrnheiser, Ferri, Petit) (*see also* p. 1221); the pneumococcus has been recovered from the pus. Mandl reports a case of hypopyon ulcer with pneumococci in the hypopyon; this observation suggests that it may have been metastatic. Considering the causation of ulcus serpens it is surprising that it does not occur more frequently in pneumonia; possibly the explanation is to be found in an acquired immunity. Schmidt-Rimpler describes a case of hypopyon ulcer in which pneumonia developed. Römer obtained fatal septicæmia in animals by infecting the conjunctival sac with cultures of pneumococcus. Paralysis of the third nerve (Gubler), sixth nerve (Westhoff), accommodation (Scheby-Buch), mydriasis (Sighicelli, Pasternatzky), etc., have been described.

Gubler.—Arch. gén. de Méd., 1860. Scheby-Buch.—A. f. O., xvii, 1, 1871. Jacobson.—Beziehungen der Veränderungen u. Krankheiten des Sehorgans zu Allgemeinleiden, Leipzig, 1885. Rampoldi.—Ann di Ott., xv, 1886; xxiii, 1895. Herrnheiser.—K. M. f. A., xxx, 1892. Westhoff.—C. f. A., xix, 1895. Ferri.—Ann. di Ott., xxvi, 1897. Fraenkel.—A. f. O., xlviii, 1899. Mandl.—Wiener med. Woch., 1899. Römer.—Z. f. Hygiene, xxxii, 1899. Schmidt-Rimpler.—Die Erkrankungen, etc., Wien, 1905. Wopfner.—K. M. f. A., xliv, 1906.

Purulent lung affections may give rise to metastatic ophthalmia, bronchitis with episcleral abscess (*S. aureus*) and iridochoroiditis (Adler), broncho-pneumonia with endophthalmitis (staphyloccoci and streptococci, no pneumococci) (Despagnet). Empyema may cause neuro-retinitis (Handford), cerebral symptoms with amaurosis (de Cérenville).

Chronic lung affections other than tubercle, etc., may cause symptoms of implication of the sympathetic nerve (Rampoldi, Pasternatzky).

Malignant tumours of the lungs may give rise to orbital or ocular metastases (*v.* Vol. II., p. 533). Similar tumours of the mediastinum may also cause the same result (de Schweinitz and Meiggs, Sänger).

ADLER.—Wiener med. Presse, 1889. DESPAGNET.—Rec. d'O., 1896. HANDFORD.—Trans. Clin. Soc., 1888. DE CÉRENVILLE.—Rec. méd. de la Suisse romande, 1888. RAMPOLDI.—Ann. di Ott., xv, 1886; xxiii, 1895. PASTERNATZKY.—Wratsch, 1886. DE SCHWEINITZ AND MEIGGS.—Am Jl. of Med. Sc., 1894. SÄNGER.—Deutsche med. Woch., 1888.

DISEASES OF THE CIRCULATORY SYSTEM.

v. Schultén has shown that the blood-pressure in the ophthalmic artery is only a few mm. Hg. below that of the carotid. Considering the very different blood supply of the eye it would be unwise to apply this result to man. Nevertheless, we may be sure that the arterial pressure is far above the normal intra-ocular pressure. It would not be surprising, therefore, if the pulse wave was transmitted to the central artery of the retina, and could be observed ophthalmoscopically. This, however, is seldom if ever the case under normal circumstances. This is doubtless due to two causes: (1) The intra-ocular pressure damps the pulsation, and the increase of pressure which accompanies each pulsation is spread over the whole volume of the contents of the globe, and is transmitted to the plastic sclerotic; (2) such pulsations as survive this damping effect are too small to be observed in these small vessels by ordinary ophthalmoscopic magnification.

Two types of *arterial pulsation* occur pathologically: (1) A true pulse wave, accompanied by locomotion of the vessels; (2) an intermittent flow of blood, or pressure pulse. In the latter, the arteries fill with blood only with the heart beats, being empty between them; it is only visible upon the disc. This type of pulsation is a pure pressure phenomenon, and is caused by any considerable increase of intra-ocular tension with normal or lowered blood-pressure, as in glaucoma, or by any considerable diminution of blood-pressure with normal intra-ocular tension, as in syncope, orbital tumours, etc. The true arterial pulse occurs in cases of aortic regurgitation (Quincke) or aneurysm, in Graves' disease, etc.; it is not confined to the optic disc. It is equally a pressure phenomenon, but the differences of pressure are smaller.

Capillary pulsation is seen only in aortic regurgitation as a systolic reddening and diastolic paling of the disc (Becker, Schmidt-Rimpler, Quincke).

Venous pulsation occurs in three forms: (1) The normal negative venous pulse; (2) the positive venous pulse; (3) the transmitted centripetal venous pulse.

The normal venous pulse occurs in 70 to 80 per cent. of people (Lang and Barrett); it is negative or diastolic, *i. e.* the veins are narrowed when the arteries are dilated. It is generally absent in lower animals. It can only be seen upon and near the disc. It is probably caused in exactly the same manner that the venous pulse is caused in the intra-cranial sinuses. There "the brain, as is shown by the cerebral pressure gauge, is lifted up by the stroke of the arteries at its base, and is thrown against the cerebral veins" (Hill). In the eye the incompressible vitreous corresponds to the brain, but the sclerotic will also yield slightly to the shock, and so the pulsation in the veins will be less marked. In this manner each systole of the heart pro-

duces a dilatation of the central artery, which is transmitted through the vitreous to the central veins, leading to a constriction, with diminished outflow of blood. This is the classical explanation of Donders. The constriction manifests itself first at the disc, and hence first affects the termination of the veins there, damming back the blood in the smaller veins. Moreover, this is the spot where the venous pressure is lowest, and it therefore responds most readily and most completely. The increased intra-ocular tension induced by the cardiac systole does not act equally and simultaneously upon the whole of the veins, leading to constriction and more rapid outflow of blood, as was suggested by Coccius. But there are other important factors which must be taken into account. One of these is direct pressure of the artery upon the vein during their course in the optic nerve, before entering the eye (Jäger). Moreover, the response of the wall of the eye to the increased pressure will be most marked at the disc (Jacobi), where, although the nerve substance is incompressible, the lamina cribrosa contains much elastic tissue, and where there is a normal exit of lymph. Further, there is a tendency to a transmitted wave, caused by the high extra-vascular tension inside the globe, so that there may normally be some transmission of the pulse through the capillaries into the veins (Türk). More important in man is the relationship to the intra-cranial circulation, though this factor can be of little importance in most animals. Helfreich thinks that the venous constriction occurs before the cardiac systole, and may continue awhile during it. He attributes this to the blocking of the cavernous sinus during the systole, and its expansion during the diastole. If, however, this were the sole cause, we should expect to find actual dilatation of the veins during the systole, and not constriction. Probably Haab is right in attempting to reconcile Helfreich's theory with that of Donders. It seems most likely that what happens is this. The intra-ocular effect of the cardiac systole is constriction of the veins; at the same moment the blood is dammed back from the cavernous sinus. It cannot flow back into the eye, but the communication with the facial and other veins tends to relieve the pressure. Soon after the diastole has commenced the pressure in the orbital veins is still high, and the blood is streaming through the intra-ocular capillaries and the veins dilate.

The positive venous pulse is presystolic—systolic. It commences with the auricular and continues through the ventricular systole. It is due to tricuspid regurgitation, and is permitted by the normal insufficiency or absence of the valves of the jugular veins.

The transmitted centripetal venous pulse is an accentuation of the normal tendency of the pulse wave to progress through the capillaries into the veins, owing to the intra-ocular pressure. It is due to venous congestion with or without increased *vis a tergo* (Holz).

COCCIUS.—Ueber die Anwendung des Augenspiegels, Leipzig, 1853. v. GRAEFE.—A. f. O., i, 1, 1854. JAEGER.—Wiener med. Woch., 1854. DONDERS.—A. f. O., i, 2, 1855. O. BECKER.—K. M. f. A., ix, 1871. FITZGERALD.—Brit. Med. Jl., 1871. STEPHEN MACKENZIE.—Med. Times and Gaz., 1875. JACOBI.—A. f. O., xxii, 1, 1876. SCHOEN.—K. M. f. A., xix, 1881. HELFREICH.—A. f. O., xxviii, 3, 1882. v. SCHULTÉN.—A. f. O., xxx, 3, 1884. LANG AND BARRETT.—R. L. O. H. Rep., xii, 1888. HOLZ.—Berliner klin. Woch., 1889. RAEHLMANN.—K. M. f. A., xxviii, 1890. MICHAELSEN.—C. f. A., xv, 1891. HALE.

—K. M. f. A., xxxv, 1897. Haab.—Atlas ; in Norris and Oliver's System, iv. Türk.—
A. f. O., xlviii, 3, 1899. Parsons.—The Ocular Circulation, London, 1903.

The degree of distension of the retinal vessels varies considerably
under normal circumstances, and greatly under pathological conditions.
Brief reference will be made here only to variations due to causes lying
outside the eye.   Uhthoff has examined the eye of the same side
during ligature of the common carotid artery; there was slight transitory
paling of the disc and narrowing of the retinal arteries: the transitoriness
of the effect is due to the free anastomosis of the cerebral arteries
through the circle of Willis (v. Vol. III, Chap. XVII).   Permanent dis-
turbance in such cases is caused by complications (Siegrist).   During syn-
cope there is narrowing of the arteries with or without narrowing of the
veins (Poncet, Coccius), and arterial pulsation may arise (Wadsworth).

Venous congestion is seen in its most pronounced form in congenital
disease of the heart with stenosis of the pulmonary artery and patent
foramen ovale ; there may be retinal hæmorrhages (Knapp, Leber,
Liebreich, Nagel).   Usually the arteries are also distended unduly.
General venous congestion from various causes seldom produces much
change in the retinal veins (Leber, Schlepegrell, Schmall).   Compression
of both internal jugular veins causes slight retinal venous congestion
and disappearance of venous pulsation, which recurs on deep respira-
tion (v. Michel).   The condition of the retinal vessels varies in epileptic
fits (Horner, Gowers) : so-called retinal epilepsy (Hughlings Jackson)
is probably a complicated condition in which papillitis plays a part.
The curious cases of vitreous hæmorrhage occurring in young adults
without obvious cause may be mentioned here (Eales, Abadie, Zieminski,
Hutchinson, Mayweg, Beaumont, Johnson Taylor, Friedenwald, Simon,
Fischer, Manzutto).

Virchow.—Handbuch, i, 1854. A. Graefe.—A. f. O., viii, 1, 1861. Liebreich.—
Atlas, Tafel ix, 1863. Knapp.—T. Am. O. S., 1870. Poncet.—Rev. gén. de Méd., 1870.
Horner.—K. M. f. A., xii, 1874 Hughlings Jackson.—Lancet, 1874 ; R. L. O. H. Rep.,
viii, 1874. Leber.—In G.-S., v, 1877. Wadsworth and Putnam.—T. Am. O. S., 1878.
Nettleship.—Brit. Med. Jl., 1882. Eales.—Ophth. Rev., i, 1882. Edmunds.—T. O. S.,
ii, 1882 Abadie.—Ann. d'Oc , xcy, 1886. Zieminski.—Rec. d'O., 1887. Hutchinson.—
R L. O. H. Rep., xii, 1889. Mayweg.—B. d. o. G., 1889. Nagel.—Mittheil. a. d. ophth.
Klinik in Tübingen, 1890. R. Batten.—Lancet, 1891. Beaumont.—Ophth. Rev., xi,
1892. Johnson Taylor.—Internat. Congress, Edinburgh, 1894. Friedenwald, Simon.—
C. f. A., xx, 1896. Fischer.—C. f. A., xxi, 1897. Geigel.—Münchener med. Woch.,
1897. Manzutto.—B. z. A., xxxiv, 1898. Goldzieher.—C. f. A., xxviii, 1904. Posey.—
T. Am. O. S., 1905.

**Obstruction (embolism and thrombosis) of the central artery of the
retina** was first described and diagnosed by v. Graefe (1859) ; the accuracy
of the diagnosis was proved anatomically by Schweigger.   The charac-
teristic ophthalmoscopic picture, with narrowing of the retinal vessels,
a white area at the posterior pole with a cherry-red spot at the macula,
etc., is so well known as to require no detailed description here.   In a
considerable number of cases there is disease of the heart (endocarditis,
valvular incompetence), in still more there is evidence of arterio-
sclerosis, in a few aneurysm of the carotid system ; in many no obvious
general disease is present.   The comparative rarity of the condition
may be partly accounted for by the almost rectangular origin of the

ophthalmic artery from the internal carotid and of the central artery from the ophthalmic or one of its branches (usually the fronto-nasal), in consequence of which an embolus is more likely to pass into one of the other branches.

The literature of the subject has recently been collected and examined by Coats, and he has given an admirable discussion of the pathological problems which arise.   Much of the following account is taken verbatim from his paper.   The total number of cases in which a pathological examination has been made is twenty-six.   The diagnosis of embolism, true for some cases, notably the first examined by v. Graefe, has been shown by many writers, *e. g.* Loring, Nettleship, Priestley Smith, to be untenable for other analogous clinical types.

FIG. 821.—OBSTRUCTION OF THE CENTRAL ARTERY.   × 120.

Coats, R. L. O. H. Rep., xvi, Case 1.   Showing the central vessels within the lamina cribrosa.   The artery is blocked by a granular mass.   The two branches of the vein are also occluded.

Within recent years, also, several cases have been reported from the purely pathological standpoint tending to diminish the importance of true embolism, and to raise into prominence other factors, such as endarteritis or thrombosis or a combination of the two.   This point of view has been especially upheld by Haab and his pupils, and Reimar even goes so far as to say that there is no case on record in which the pathological examination has proved with certainty that a true embolism was the cause of blindness.   This statement is much too positive, for there is no reason to doubt that embolism furnishes the best explanation of the pictures and descriptions given by Schweigger, Manz, and Marple. Coats's first case and probably Nettleship's first case also belong to the

same category, and the clinical aspects of Gowers's case make this diagnosis likely.

On the other hand, it is not to be denied that, both from the clinical and pathological standpoints, a strong case has been made out for endarteritis as the cause of obstruction in many instances—*e. g.* in the cases of Elschnig, Wagenmann, v. Michel, and Galinowsky. Thrombosis must, no doubt, be reckoned as a very important factor in these cases, but it is not a primary factor, and so far as the pathological evidence goes, does not occur as a causal agent except in association with disease of the vessel wall. A minor exception of no importance to the main question is furnished by a case of Siegrist's, in which blindness of
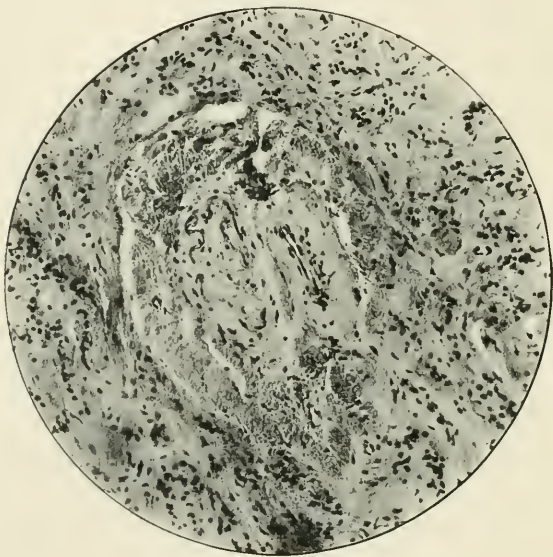
FIG. 822.—OBSTRUCTION OF THE CENTRAL ARTERY.   × 120.

Coats, R. L. O. H. Rep., xvi, Case 1.   Showing the central vessels posterior to the lamina cribrosa.   The artery, to the left, is collapsed and obliterated by proliferation of endothelium.   The vein is concentrically narrowed by thickening of its walls, not by endothelial proliferation.

one eye followed upon ligature of all the carotid arteries of the same side, and a coagulum was found in the ophthalmic and central arteries. Such a case, of course, stands in a class by itself, and need not enter into the discussion of the subject of obstruction of the central artery as ordinarily observed clinically (*cf.* Uhthoff).

Theoretically, certain other lesions might cause the same clinical picture, such as retrobulbar neuritis, spasm of the arteries, hæmorrhage within the optic nerve or in its sheath (Magnus), and tumours in the same situations ; but none of these rests on any pathological evidence.

Spasm of the arteries has the support of certain analogies, notably the fine thread-like appearance of the vessels in quinine amaurosis,

which has been supposed to be due to this cause.   Narrowing of the
retinal arteries from spasm has also been described by Raynaud in the
disease which goes under his name, but the observation has never been
repeated, and has been denied (by Panas) even for the case in which
Raynaud described it. (See Raynaud, 'Arch. générale de Médicin,' 1874;
Panas, ' Les Maladies des Yeux.' Panas was a colleague of Raynaud's,
and examined his ophthalmological cases for him.   The whole is
quoted in Reimar's papers in ' Arch. f. Augenheilk.,' vol. xxxviii, 1899,
p. 291.)   It is possible that spasm may have played a part in some
instances, but almost all the cases which have been pathologically
examined have shown changes quite sufficient to account for the

FIG. 823.—OBSTRUCTION OF THE CENTRAL ARTERY.   × 120.

Coats, R. L. O. H. Rep., xvi, Case 2. Showing the central vessels within the
lamina cribrosa.   The artery, to the left, is almost occluded by an endarteritic
nodule.   The dark granular mass is calcareous.   A small branch is blocked by
endothelial proliferation.   The lumen of the vein is encroached upon by fibrous
tissue.

symptoms without calling in its aid.   Records of spasm of retinal vessels
observed ophthalmoscopically are always to be received with caution,
as the condition is easily counterfeited when the vessels are badly filled
from any cause.

An interesting clinical point in several cases is the preservation for
a time of a little vision in the temporal part of a field, although the
picture is that of complete obstruction.   This is by no means un-
common in recorded cases, apart altogether from the occurrence of
unblocked cilio-retinal or macular arteries.   The preserved area is
almost without exception on the temporal side of the field—on the

nasal side of the fovea therefore. Coats was unable to find a perimeter tracing showing its exact position; indeed, perimetry would be difficult to carry out in these cases, since the patient has lost central vision. But an observation of Fischer's is of interest in this connection. By noting on what part of the fundus the ophthalmoscopic image of a small flame fell, he found that the patient had perception of light only in an area of about one third disc diameter around the papilla. The explanation at once suggests itself that the capillary anastomoses between the ciliary and central vessel systems in this region are capable of keeping up the nutrition of a small zone of retina in the immediate vicinity of the disc (v. Vol. III, p. 949). In some cases, however, the

FIG. 824.—OBSTRUCTION OF THE CENTRAL ARTERY.   × 120.
Coats, R. L. O. H. Rep., xvi, Case 2.   Showing the central vessels posterior to the lamina cribrosa.   The artery, to the left, is full of blood and shows no endarteritis.   The lumen of the vein is very small.

area is much more peripheral, and sufficient nutrition for perception of light is probably afforded by the choroidal circulation.

The question is much debated as to the amount of collateral circulation which may be established when the main artery is blocked. It is usually taken for granted that this is *nil*, and, indeed, for practical purposes and so far as the functions of the retina are concerned, it may be admitted that the central artery is an end artery. Yet it is a common observation that arteries which have been thread-like and almost invisible at the time of the occurrence of obstruction become after a while decidedly better filled or even tolerably well filled. Afterwards, in the course of weeks or months with advancing atrophy of the disc, they again become small and frequently sclerosed.

This well-established clinical fact has given rise to very various conjectures as to its cause. The earliest explanation (Schneller) was

that the obstruction was situated in the ophthalmic artery at the point where the central artery was given off, and that it was afterwards carried onwards so as to leave the central artery free to fill up again. While such an occurrence is theoretically possible, it must be admitted that it could only happen very exceptionally, and since the refilling of the arteries is the rule rather than the exception, the hypothesis must be discarded, more especially as no proof has been brought forward in support of it. Another supposition has been that the embolus gets broken up by the blood-pressure behind it, and the fragments scattered among the retinal arteries, but this is probably impossible without some ophthalmoscopic evidence—evidence which is usually entirely absent (cf. Hirschberg). Or, again, the theory of "incomplete embolism" has been advanced. The plug stops at a part of the artery which it does not completely fill, and the obstruction is made complete by spasm of the arterial wall. Afterwards this spasm relaxes, and the vessel may even become abnormally dilated owing to ischæmic paralysis. Hence a current is re-established. The weakness of this explanation lies in the supposition that a mass whirled along by a tolerably swift current will stop before it is brought up by the narrowness of the vessel lumen or by impinging against a bifurcation. Hence, the theory has been modified by supposing that the plug goes far enough to completely fill the vessel, but that the contraction of the arterial wall which it induces compresses and moulds it to a smaller size, so that when the spasm relaxes again a small lumen is left at the side. It seems probable, however, that secondary thrombosis would prevent re-establishment of the current. These explanations are highly theoretical, and rest on no clear pathological evidence.

Fig. 825.—Obstruction of the Central Artery. × 160.

Coats, R. L. O. H. Rep., xvi, Case 2. Showing a retinal artery, a superior branch which was seen obstructed ophthalmoscopically. The lumen is obliterated by hyaline laminæ.
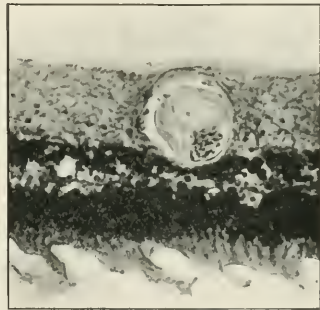
Fig. 826.—Obstruction of the Central Artery. × 160.

Coats, R. L. O. H. Rep., xvi, Case 2. Showing an inferior branch, not obstructed ophthalmoscopically. There is eccentric narrowing by endothelial proliferation.

Again, it has been supposed that if the obstructing mass were angular and hard—as, for instance, a piece of calcareous cardiac valve—it might lodge in the vessel but still allow some blood to flow past it. But it can hardly be doubted that in this case secondary thrombosis would soon make the obstruction complete. Canalisation of a plastic embolus or of a thrombus is also a possible means of refilling of the retinal arteries. Its occurrence has not been proved in the artery,

though it may occur in the vein (Coats, Sidler-Huguenin) (*vide infra*). In the cases now under consideration, however, the time which elapses before some circulation is re-established is usually much too short to allow of such an explanation. Nettleship has found that after complete obstruction of the central artery with total loss of sight, some re-establishment of the circulation (as tested by the possibility of producing pulsation in the arteries and by their refilling after emptying) may take place within two and a half hours.

There remains the possibility that the refilling of the vessels is due to the re-establishment of a small collateral circulation. This also cannot be said to be proved for all cases, though there is evidence in its favour. The objection has been raised that if a collateral circulation is formed in this manner, it should go on enlarging until the vessels have reached their normal size, instead of which the vessels after a while again become small and sclerosed. But the amount of collateral circulation which the anastomoses around the nerve head are capable of feeding must be small, since the vessels are of little more than capillary calibre, and are, therefore, probably not capable of indefinite distension, especially on the occurrence of a sudden block. It is a sufficiently well-known pathological principle that a vessel wall tends to adjust itself to the amount of blood flowing through its lumen, not only by partial collapse, but also by thickening. Thus, when the central vein is blocked, not only is the vessel collapsed and with a small lumen higher up the nerve, but its walls are enormously thickened by increase of the connective-tissue elements in them. This thickening of the wall decreases as the lumen of the vein gradually becomes better filled by the accession of collaterals. It cannot be a compensatory thickening to meet heightened blood-pressure, since the pressure must be greatly lowered just above the block ; it must, therefore, be due simply to adjustment of the vein wall to the small quantity of blood in it. The same process of vascular thickening occurs when the central artery is blocked, both in the retinal arteries and in the retinal and central veins (Fig. 826). This is probably the reason for the final smallness of the vessels, even in those cases which have refilled fairly well after the primary block. The pathogical history of a case might be divided into three stages as follows : (1) At first the arteries are thread-like from more or less sudden cutting off of the blood-stream. (2) Later there is some refilling from the establishment of a collateral circulation—we are looking with the ophthalmoscope at a small stream in more or less normal arteries. It is to be remembered that the size of arteries ophthalmoscopically is not necessarily an indication of the amount of blood flowing through them. The velocity of the circulation must also be taken into account, and this cannot be noted unless it falls very far below the normal and indicates itself by visible streaming or beading in the vessels. It is possible, therefore, to have arteries of fair size yet conducting only a comparatively small stream of blood. (3) The last stage is that of the above-mentioned fibrosis of the vessel wall—we are looking with the ophthalmoscope at a small stream in thick-walled arteries, and this corresponds with the final stage of

thread-like sclerosed arteries associated with optic atrophy in old cases of obstruction.

It is probable, however, that the freedom of the collateral circulation is subject to individual variation associated with corresponding differences in the clinical course of the case. There are several recorded instances (Schneller, Fischer, Wagenmann, Story) in which, after the picture of complete obstruction, there has been considerable recovery of sight, while on the contrary other cases have never shown any perception of light from the beginning. The usual explanation of these cases with recovery is similar to that given for cases with prodromal obscurations, viz. there is narrowing of the lumen of the central artery from endarteritis, and if the general blood-pressure falls from any cause—not necessarily low enough to cause fainting or cerebral symptoms—it may be insufficient to force the blood past the narrowed part, and the picture of obstruction of the central artery is produced. If, however, within a short time the pressure rises again, the blood may force a passage, and provided that the retina has not been too long anæmic, some vision may still be saved. This is no doubt the explanation of many, perhaps most of these cases, but it is also possible that in some instances the freedom of the collateral circulation and the rapidity with which it can be established may play some part in the process. The good effects of massage in some cases and its entire inutility in others may also be related to the freedom of the collateral circulation.

The exact position of the block, too, as Nettleship has remarked, would probably have considerable influence. If it were extremely far forward, on the inner side of the lamina cribrosa and just at the bifurcation of the vessel, the anastomoses would be shut out from participation because the block would be beyond them; but in this case it might happen that a macular branch was given off from the main artery central to the obstruction, which would preserve some vision by another mechanism similar to what occurs when a cilio-retinal artery is present. It is wrong to attribute the preservation of a small triangular field on the temporal side of the disc in every instance to a true cilio-retinal vessel. In many cases this has been the cause (Nettleship, Lang, etc.), but in others the unblocked twig has undoubtedly been a branch of the central artery itself coming off higher than the obstructed portion (Hirschberg, etc.). On the other hand, if the block lies within or central to the lamina cribrosa the anastomoses of the nerve head have full play. It seems possible that if the block is fairly far forward the superficial portions of these anastomoses will be especially dilated, and this may account for certain cases in which an anastomosis has been visible ophthalmoscopically in the form of fine networks around the papilla. Such networks are sufficiently common in cases of obstruction of veins, but after blocking of the artery they are very rare. There are only three in the literature—two by Nettleship, and one recently by Gonin. These cases furnish strong evidence that some degree of collateral circulation is possible after blocking of the main artery. A case reported by Lawson in which the arteries, which were fine and thread-like on the disc, suddenly swelled out to

their normal size as they crossed the edge, also points in the same direction.

Further and perhaps even better proof is furnished by another class of cases—those in which the central artery has been divided with the optic nerve in optico-ciliary neurotomy or for the removal of tumours of the nerve (v. p. 1184).

Again, the central vein is usually regarded as an "end" vein—a vein the obstruction of which is not compensated by a collateral circulation, and clinically it undoubtedly is so, its blocking being followed by the well-known ophthalmoscopic picture and almost always resulting in permanent damage to the retina. Yet microscopically there is evidence of collateral channels; the vein does not remain collapsed above the obstruction, but a blood-filled lumen is re-established by the accession of small vessels from the trabeculæ. A close analogy may be drawn between what occurs in the vein and what occurs in the artery. In each case on blocking, a collateral circulation is re-established, which, however, in almost all cases occurs too late and is insufficient in amount to restore function. In certain instances, however, as has been seen, some function *is* restored after complete blocking of the artery, and Coats has seen a case of typical thrombosis of the central vein in a young person in which all the hæmorrhages cleared up and the vision finally was $\frac{6}{6}$.

The little hæmorrhages which are not infrequently seen near the disc somewhat late in obstruction cases are most probably the result of the rupture of capillary vessels under the strain of dilatation in the attempt to establish the collateral circulation.

There appears, therefore, to be a considerable body of evidence in favour of the occurrence of a collateral circulation when the main artery is blocked.

It has long been considered a point against the diagnosis of true embolism if the patient gives a history of having had previous temporary obscurations of sight before the final attack of blindness, for it is evidently difficult to believe that large numbers of emboli could chance to enter the central artery, or that if they did the obscurations would be temporary and not permanent. More especially is this the case when one eye only is affected over a prolonged period. Nor does thrombosis offer a better solution of the problem, for there is no reason to think that thrombi could appear and disappear in the manner necessary. It is also difficult to believe that embolism is the correct explanation where both eyes are affected (Jessop, Olaf Page, Treacher Collins, etc.), and especially if this occurs simultaneously or nearly so (van Duyse, Knapp, Loring, Haase, etc.). In many of these cases also there has been recovery in one eye but not in the other, which seems incompatible with both embolism and thrombosis. The objection seems to have been first stated by Loring, and both he and Nettleship have founded the argument upon it that in these cases there is probably local disease of the vessel wall rendering the retinal circulation liable to temporary interruption from comparatively slight causes — slight lowering of arterial pressure (in sleep for instance), venous stasis from exertion, bending, etc. Coats's second case may be looked upon as

furnishing the pathological proof of such a supposition.   It needs but a glance at Fig. 823 to see how easily the retinal circulation might be cut off by some such slight cause, and how if this were speedily removed it might be restored again, but if not, how the block might well become permanent with or without thrombosis.   Or even if it did not become permanent the retinal elements might be too much damaged to resume their functions when the circulation was restored.   Two main factors probably go to the determination of these prodromal attacks, local arterial disease and diminished blood-pressure.   In most cases the former is of chief importance and the latter secondary.   But in some cases this relation is reversed.   Thus in a case of Priestley Smith's the obscurations were associated with fainting attacks, due to the pain caused by vaginal douching for an ovaritis.   One eye had already become permanently blind during one of these attacks, and the douching being continued, repeated obscurations were occurring in the other, associated with faintings.   On discontinuing the douchings and removing the offending ovaries both ceased permanently.   In this case Priestley Smith's explanation is probably the correct one, that the obscurations were due to cardiac inhibition from the pain, with slowing of the cerebral and ocular circulations, while the permanent blindness of the left was caused by thrombosis of the central artery in that eye. Yet this association of obscurations with other signs of deficient circulation is distinctly exceptional in recorded cases, and it is probable that in most instances the chief factor is local arterial disease, a cause which Priestley Smith also mentions in the same paper.   The only case in which the fundus was seen *during* one of these prodromal attacks is reported by Wagenmann, 'Arch. f. Ophth.,' vol. xliii, 1897, pt. 2, p. 219.   He found that the vessels were small and thread-like, and did not pulsate as in ordinary obstruction cases, but quickly refilled with the return of vision.   This case ended in an attack which did not pass off, permanent blindness resulting.

A large mass of evidence has already accumulated to show that of all parts of the ocular circulation the part in the immediate vicinity of the lamina cribrosa is that most liable to be affected by vascular disease in any form.   This is undoubtedly the case with regard to the vein (*vide infra*).   With regard to obstruction of the central artery the fact is no less striking, and this is so whether the obstruction be a true embolus, endarteritis, or endarteritis combined with thrombosis.   From the literature also the cases of Schweigger, Nettleship, Manz, Marple and Coats furnish examples of true embolism in this situation ; those of Galinowsky, and (probably) Nuel, Wagenmann, and Schnabel and Sachs examples of endarteritis ; that of Ridley (probably) of thrombosis. It is to be noted that this list includes all the best reported cases, and that in several of the others no obstruction at all was found (Hirschberg, Loring, Popp) probably because the lamina was not cut transversely, while in others again the descriptions are not satisfactory, and it is probable that secondary changes have been taken for primary.   It will be evident on looking at Fig. 822 that the changes shown there could easily be taken for the primary obstruction, considering that the eye was obtained three and a half years after the plugging.   Indeed

this mistake was very nearly made in that case, for the calcareous mass had broken out of most of the sections and was all but overlooked. The case of Galinowsky is especially interesting, for it is almost exactly the same as Coats's second case. Just above the lamina there was a patch of marked endarteritis with a calcareous nodule in it. The case differed only in that the lumen was completely blocked, and clinically there was the picture of complete obstruction and not of branch obstruction as in the present instance.

What is the explanation of the fact that the lamina cribrosa is the seat of election for these various processes ? Many factors probably come into account and act in different degrees in different cases. In the first place the artery becomes narrowed as it passes through the lamina. In a normal eye excised for orbital tumour the central artery measured 145 μ at the lamina cribrosa 166 μ a millimetre above it. This is a sufficient reason why emboli of a size to enter the central artery at all should become arrested at that point, but, in addition, it furnishes a cause for processes of endarteritis and thrombosis by increasing friction and causing eddies. Then again, just beyond the lamina the current meets with increased resistance from two factors—the bifurcation of the main trunk and the intra-ocular pressure. These reasons concern the artery alone, but in addition Haab has suggested that the ocular movements have something to do with the occurrence of vascular disease in this situation. The globe is to some extent anchored by the optic nerve, so that during its movements there may be a degree of pulling and dragging just where the nerve is inserted into the eyeball, although this is partly allowed for by the sinuosity of the nerve in its orbital course and by the fact that it is a little longer than the distance from foramen opticum to globe. This factor will act chiefly on the thin-walled and collapsible vein. It is possible also that the lamina cribrosa, which is chiefly composed of elastic tissue, exercises some elastic pressure on the vessels as they pass through it, and that the vessels are, therefore, less capable of compensatory dilatation in this situation. In addition it is certain that the connective tissue around the central vessels is more dense in the region of the lamina cribrosa than elsewhere—as if for protection of the vessels from some outside pressure (Figs. 823–4).

The next question which must engage attention is the connection, if any, between obstruction of the central artery and the subsequent occurrence of glaucoma. In the case of thrombosis of the central vein such a connection has been abundantly proved, and most of the pathological material has been obtained in consequence of this sequence of events (vide infra). With regard to obstruction of the artery, on the other hand, while several cases have been reported in which glaucoma followed, the connection is much less certain, and a large proportion of the eyes pathologically examined has been obtained from the post-mortem room. In all, glaucoma has been observed some eight times. It followed within four days of the use of a mydriatic in one case (Ridley) ; a mydriatic had been used in two other cases (Nettleship), but whether it had anything to do with the onset of glaucoma or not is doubtful. In another case there were definite signs

of iritis (Galinowsky); in Loring's case there were retinal and vitreous hæmorrhages.   In a case of Knapp's, which presents many points of resemblance to Coats's second case, the patient suffered from diabetes and an obstruction of the central artery occurred in one eye; later hæmorrhagic iridochoroiditis developed and glaucoma followed probably as a consequence.   Thus there are left two (Manz, Marple), or counting Nettleship's two cases, four cases in which obstruction of the artery and glaucoma occurred in simple association.   Is this number higher than might be accounted for by coincidence, or by the obstruction and the glaucoma being due to a common cause, arterio-sclerosis, but not dependent the one on the other ?   In 1891 Fischer had collected over 150 cases of obstruction, and the number of *reported* cases cannot now number less than 200.   This would give a percentage of 1 or 2, but in reality such a calculation is of no value whatever, for the clinical picture of obstruction is so well worked out now that only cases presenting some new or peculiar feature are likely to be reported.   On the other hand, cases complicated with glaucoma will probably be published both on account of the rarity of the combination, and because when it occurs it is not unlikely to lead to obtaining the eye for pathological examination.   It is probable, therefore, that these eight cases are all, or nearly all, that have occurred since the clinical characters of obstruction were well known, so that the condition must be one of great rarity—a supposition which accords well with clinical experience.   The combination with glaucoma has not been confined to one type of case; Nettleship's, Manz's and Marple's were probably true emboli, Ridley's thrombosis, and Galinowski's endarteritis.

In Coats's two cases glaucoma was present.   In the first it had evidently nothing to do with the embolus, but resulted entirely from changes in the anterior part of the eye.   In the second case also it was probably not connected with the obstruction of the retinal arteries.   There was much hæmorrhage in the root of the iris and among the spaces of Fontana, probably in consequence of vascular disease the result of diabetes, or from the abnormal constitution of the blood from the same cause.   The glaucoma was therefore most probably to be referred to interference with the drainage channels.   Knapp's case mentioned above is another example of the association of glaucoma with diabetes and hæmorrhagic iridochoroiditis.   Hirschberg has also reported a case of glaucoma in a diabetic; it followed upon a hæmorrhagic retinitis.   It is difficult to suggest any reason why glaucoma should be caused by a block on the central artery, for such a block will not give rise to any back pressure within the eye or damming back of lymph or blood.   There will not, probably, be any alteration in the chemical constitution of the fluids in the vitreous or anterior chamber, and any explanation depending on changes in the characters of the fluids to be drained off is therefore improbable.   In glaucoma following upon thrombosis of the central vein there are many obscure points, but at least there is obstruction of outflow, and some such alteration in the chemical characters of the fluids is conceivable.   The evidence, therefore, does not seem at present to be sufficient to decide whether there is any true causal connection between obstruction of the central

artery and glaucoma. On the whole the probabilities are against it, but the decision must be left to future research.

Coats has summarised the cases of obstruction of the central artery which have been subjected to pathological examination. The condition of the retina has not been mentioned, for in all the cases in which it has been described it has been the same, viz. atrophy of the nerve-fibre and ganglion cell layers, with preservation of the outer layers which receive .their nourishment from the chorio-capillaris of the choroid. It has been asserted, with probability, on *à priori* grounds that the retinal haze seen soon after the occurrence of the obstruction is not due to œdema as is frequently assumed, but to parenchymatous degeneration or coagulation necrosis of the inner layers of the retina supplied by the central artery. No case seems to have been examined early enough to prove this definitely, and œdema has certainly been found in later stages by several authors (Hirschberg, Nettleship, Nuel, Ridley, Manz).

A few explanatory and critical remarks with regard to the cases on the list may be permitted. Schweigger's is the case originally observed by v. Graefe on which the first description of "embolism of the central artery" was founded. From the picture given it seems to be a true case of embolism altogether similar to Coats's first case. Sichel's was not a typical case clinically—there was a large macular hæmorrhage—and the significance of the pathological lesion found is also doubtful. Most probably it was a nodule of endarteritis, possibly calcareous, and analogous to Coats's second and Galinowsky's cases. In Loring's case no obstruction was found, but it seems probable from the clinical history that one must have been present which was overlooked. The region of the lamina cribrosa was cut longitudinally, not transversely. In Nettleship's first case there was undoubtedly an organising plug in the central artery, but whether an embolus or a thrombus it would be difficult to say from the pathological examination alone. The clinical history (aortic disease, sudden blindness without prodromata, etc.) makes an embolus more probable. His second case (Nettleship and Watson) is more doubtful, as he himself admits. His third case (Nettleship and Lawford) seems to be one of endarteritis (? with thrombosis) the result of diabetes, and presents many points of resemblance to Coats's second case. The obstruction was found in the case of Schmidt-Rimpler, but neither the description nor the picture enables one to come to any certain conclusion as to the nature of the lesion. There is no picture given of Priestley Smith's case, and the description is very brief, so that beyond the fact that the artery was occluded nothing certain can be affirmed. Gowers states that the masses found by him in the central artery were probably not the original obstruction, since the vessel was collapsed above (proximal to) them. He believes that the true embolus had lodged at the origin of central artery from the ophthalmic, beyond the portion of nerve included in the sections. The association of the obstruction with emboli in the middle cerebral artery and in the kidneys and spleen makes it probable however that it was a true embolus. In Popp's case nothing was found, and it must be supposed that the obstruction was overlooked (lamina cut longitu-

dinally). It is impossible to believe with Elschnig that an embolus or thrombus can be absorbed and leave no trace. The same remarks apply to Hirschberg's case. The case of Schnabel and Sachs was most likely one of endarteritis—small peripheral lumen clothed with endothelium. No picture is given. Manz's case seems to be a typical true embolus closely similar to Schweigger's, Marple's, and Coats's Case 1. A very excellent picture will be found in this paper. In Elschnig's the pictures clearly show that the lesions described as emboli are endarteritis. There is the typical eccentric endothelium-lined lumen and thickening mainly confined to the inner coat. Wagenmann's case is probably of the same nature, but not so certainly. Marple's is another case of typical embolus. In Ridley's paper the picture is far from clear, but from the description it seems sufficiently probable that the lesion found was a thrombus—whether a secondary thrombus or the true cause of the symptoms must be doubtful. The lamina was cut longitudinally. In Nuel's case the real obstruction seems to have been found. It is an organising mass, presumably a plastic embolus or thrombus. Cardiac disease was present. Siegrist's was undoubtedly a case of thrombosis spreading from the carotid after ligature, but as before stated it belongs to a class different from all the others mentioned. v. Michel's case is a typical one of endarteritis and thrombosis, much like Coats's Case 2 and Galinowsky's case. Welt's case is altogether anomalous, both clinically and pathologically. It does not belong to the embolism group, but to the group of cases of blindness following copious hæmorrhage. Leaving out of account the somewhat remarkable fact that detachments of the retina disappeared within a few days in *both* eyes, there remains the explanation of a blindness of some three weeks' duration by a thrombus at most a few days old. Still more remarkable is the statement that while the ophthalmoscopic picture was similar in the two eyes, the central artery was obstructed in one and the vein in the other. The pictures are not more convincing than the descriptions, and the case must be altogether rejected. Galinowsky's case is an excellent example of a nodule of endarteritis in the region of the lamina, and is well illustrated. It bears a very close resemblance to Coats's Case 2, except that the obstruction was complete. The pictures given in Hofmann's paper show nothing but an artery cut obliquely. The similar condition found in the other eye—in which there were no signs of arterial obstruction—is thus easily explained. It is impossible to accept Gonin's explanation of his case. He found a granular mass in the artery indeed, but believed it to be a thrombus secondary to a block in the vein. The consequences of obstruction of the central vein are, however, well known, and bear no resemblance to those of obstruction of the artery. In the same paper Gonin gives a case of severe hæmorrhagic retinitis in which an obstruction was found in the artery. To accept these two cases would be to upset innumerable other authentic records, so that an error of observation must be supposed.

Certain other cases might be quoted from the literature which have a more or less direct bearing on the subject without being in all

respects typical. Thus Rothmund and Eversbusch state, without giving details, that in a case of " embolism of the central artery " the diagnosis was strengthened at the autopsy by the discovery of a thrombus in the right carotid, middle cerebral, and ophthalmic arteries. Apparently there was no microscopic examination of the central artery. Again, a case of Friedmann's lends support to explanations depending upon endarteritis, or endarteritis with thrombosis. A man, æt. 52, without any cardiac or renal lesion, but with rigid radials, became affected with obstruction of the right central artery. A year later he developed spastic paralysis, and two and a half years after this he died of apoplexy. The whole system of basal arteries, down to the smallest twigs, showed obliterative endarteritis, and although the central artery was not microscopically examined, it seems probable that the blindness was due to a similar condition in it probably associated with thrombosis.

The general result of the examination of the literature, therefore, would seem to be that ample proof has been furnished that obstruction of the central artery may be due to one of three causes : (1) embolus ; (2) endarteritis alone ; (3) endarteritis with thrombosis. Thrombosis without endarteritis probably only occurs in exceptional cases, as where the carotids have been ligatured. There is at present no pathological proof that the obstruction may be caused by hæmorrhage into the nerve or nerve sheath, or by spasm apart from endarteritis.

As regards the exact condition of the blood-vessels, the arteries are extremely small, but they still contain blood. The veins are narrow at the disc, and full or abnormally distended towards the periphery. The intra-ocular tension is not appreciably altered, owing to the intact ciliary circulation, and this accounts for the absence of the formation of infarcts, such as occur when " end arteries " are blocked in other parts of the body. As mentioned above, when only a small arterial branch has been blocked it is not easy to understand why the intra-ocular tension does not actually force the blood out of the veins, especially when we consider that the pressure in the orbital veins must be considerably less. This is shown by the fact that in dogs the cerebral venous pressure is normally about 100 to 130 mm. H_2O (Hill), i. e. about 10 mm. Hg., whilst the intra-ocular tension is from 20 to 30 mm. Hg. Probably in most cases of embolism due to mitral stenosis, etc. the general venous pressure, and with it the cerebral venous pressure, is raised ; but the ocular venous pressure, and with it the capillary and intra-ocular tension, will also be raised, so that this fact affords no explanation. The elasticity of the arteries tending to keep them open, and so produce a negative pressure, must be extremely small, and well below the intra-ocular tension. The fall in tension at each cardiac diastole will also tend to draw the blood back into the eye, but this effect must also be negligible. On the other hand, it is surprising that no pulsation of the veins is noticeable due to this cause. The anastomosis with the ciliary system at the disc would tend to fill the veins from the centre towards the periphery, so this cannot be the full explanation. The most potent factor is probably the fact that the

ocular venous tension is lowest at the disc, so that the veins are stopped here first by the extra-vascular pressure, and the blood is dammed back.

Both the constriction of the veins here in embolism and the normal venous pulsation tend to point to some definite obstruction at this spot, impeding the exit of blood, and this is most likely to be found in the elasticity of the lamina cribrosa, which acts under such advantageous conditions in the narrow scleral canal.

The question is nearly allied to the condition in which the central vessels are cut across, as in optico-ciliary neurotomy, orbital wounds, etc. (v. p. 1184).

v. Graefe.—A. f. O., v, 2, 1859. Schneller.—A. f. O., viii, 1, 1861. Schweigger.—Vorlesungen, 1864. Haase.—A. f. A., x, 1868. Schirmer.—K. M. f. A., vi, 1868; in G.-S., xi, 1, 1904. Knapp.—A. f. A., iii, 1869; x, 1881. Mauthner.—Med. Jahrb. d. Wiener Aerzte, 1873; Allg. Wiener Med. Zeitung, 1873. Landesberg.—A. f. A., iv, 1874. Magnus.—Die Sehnervenblutungen, Leipzig, 1874. Loring, Olaf Page.—Am. Jl. of Med. Sc., 1874. Schmidt-Rimpler.—A. f. O , xx, 2, 1874; Die Erkrankungen, etc., Wien, 1905. Gowers.—Lancet, 1875. Leber.—In G.-S., v, 1877. Loewenstein.—K. M. f. A., xvi, 1878. Nettleship.—Brit. Med. Jl., 1879; R. L. O. H. Rep., xi, 1887; Festschrift für v. Helmholtz, 1891. Nettleship and Lawford.—T. O. S., ii, 1882. Rothmund and Eversbusch.—Mitt. a. d. Univ. Augenanstalt zu München, 1882. Hirschberg.—C. f. A., viii, 1884. Priestley Smith.—Ophth. Rev., iii, 1884. Schnalbel and Sachs—A. f. A., xv, 1885. Kamocki.—A. f. A., xvii, 1887. Fischer.—Ueber Embolie der Arteria centralis retinæ, Leipzig, 1891. Kern.—Dissertation, Zürich, 1892. Elschnig.—A. f. O., xxxix, 4, 1893. Uhthoff.—A. f. O., xxxix, 1, 1893; Internat. Congress, Rome, 1894. Wagenmann.—A. f. O., xliv, 2, 1897. Treacher Collins.—T. O. S., xvii, 1897. Seydel.—Z. f. A., ii, 1897. v. Michel, Siegrist.—B. d. o. G., 1898. Lawson.—T. O. S., xviii, 1898. Graefe.—Deutsche med. Woch., 1899. Lang.—T. O. S., xix, 1899. Reimar.—A. f. A., xxxviii, 1899 (Bibliography). Story.—T. O. S., xix, 1899. Welt.—A. f. A., xli, 1900. Siegrist.—Die Gefahren der Ligatur der grossen Halsschlagadern, Leipzig, 1900. Haab.—In Norris and Oliver, iv, 1900. Thompson, Jessop.—T. O. S., xx, 1900. Friedmann.—Deutsche Z. f. Nervenheilk., xvi, 1900. v. Duyse.—A. d'O., xxii, 1902. Parsons.—The Ocular Circulation, London, 1903. Sidler-Huguenin.—A. f. A., li, 1904. Gonin.—A. d'O., xxv, 1905. Harms.—A. f. O., lxi, 1905. Velhagen.—K. M. f. A., xliii, 1905. *Coats.—R. L. O. H. Rep., xvi, 3, 1905. Fejér, Hirschberg.—C. f. A., xxx, 1906. Gross.—A. f. A., lvi, 1906. Zentmayer.—Ann. of O., 1906. *Shiba.— A. f. O., lxii, 1906. Gräfenberg.—A. f. A., liv, 1906. Schwitzer.—Z. f. A., xvi, 1906. Verhoeff.—Ophth. Rec., 1906.

Embolism or circumscribed thrombosis of the posterior ciliary vessels, other than septic or neoplastic, is probably devoid of serious results owing to the free anastomosis, though cases have been described (Knapp, Siegrist).

Knapp.—A. f. O , xiv, 1, 1868. Siegrist.—B. d. o. G., 1898.

General arterio=sclerosis manifests itself by changes in the retinal vessels—often the first observable sign. The histological changes have already been described (v. Vol. II, p. 587). The ophthalmoscopic changes have been well described by Raehlmann, Marcus Gunn and others. Retinal hæmorrhages are often due to this cause and are of serious import as regards life (Hasket Derby). Miliary aneurysms are rare (Sous, Magnus—arterio-venous, Litten, Fuchs—arterio-venous, Benson, Schleich, Story, Schmall, Raehlmann, Seydel—arterio-venous). The arterio-venous aneurysms are usually traumatic; they do not lead
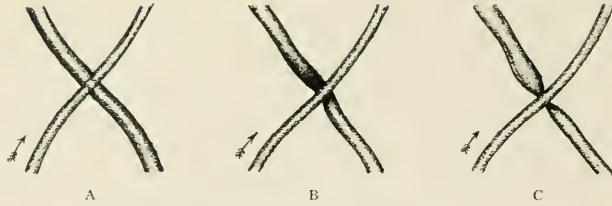
FIG. 827.—VASCULAR DEGENERATION.

Marcus Gunn, T. O. S., xviii. Showing diagrammatically appearances pre-
sented by a retinal vein where crossed by an artery. A. In health, the underlying
vein dimly traceable beneath the artery. B. In early stage of arterio-sclerosis, the
vein somewhat displaced in direction of arterial circulation, and its blood-flow
slightly obstructed. C. In advanced stage of arterio-sclerosis, the vein greatly
narrowed where crossed and distended on the peripheral side.
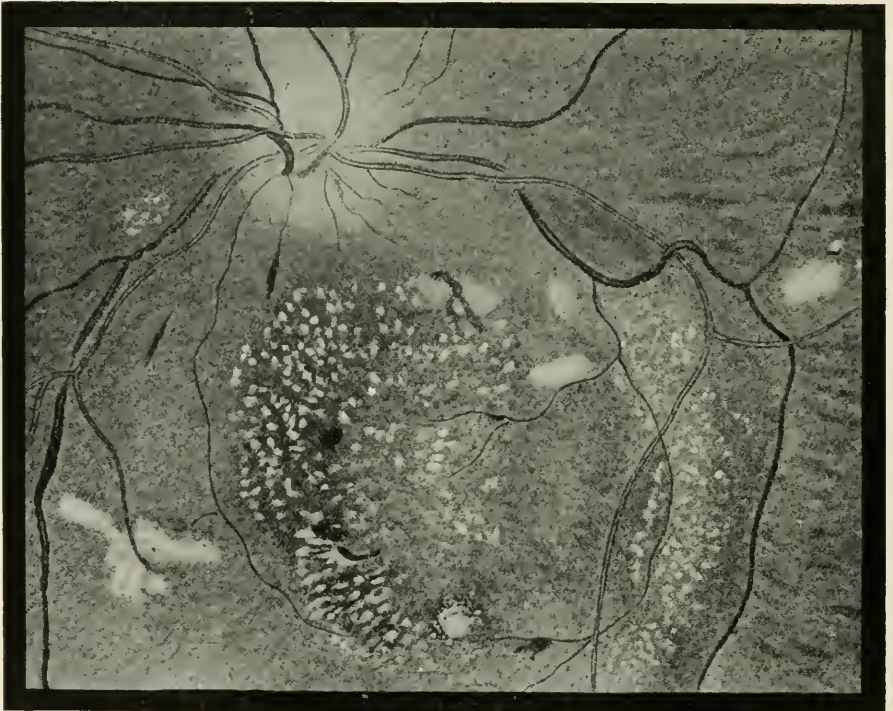
FIG. 828.—VASCULAR DEGENERATION.

Percy Flemming, T. O. S., xxiv. Showing " silver wire " arteries, obstruction
of circulation in veins, especially upper temporal vein, hæmorrhages, and white
exudates. From a patient with albuminuria who died of apoplexy two months
later.

FIG. 829.—VASCULAR DEGENERATION.

Parsons, Diseases of the Eye. "Silver wire" arteries, degeneration of the walls of a vein, and white spots of degeneration. From a case of W. T. Lister.
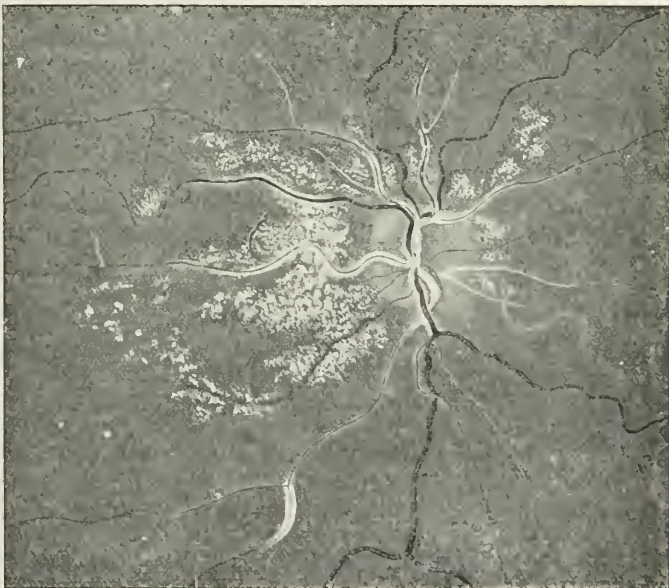
FIG. 830.—VASCULAR DEGENERATION.

Parsons, Diseases of the Eye. Perivascular changes in the retinal vessels and white spots of degeneration. From a case of J. B. Lawford.

to great distension owing to the external support afforded by the intra-ocular pressure.

EDMUNDS.—R. L. O. H. Rep., x, 1880. HIRSCHBERG.—C. f. A., vi, 1882. FÜRSTNER.—Deutsches A. f. klin. Med., xxx, 1882. RAEHLMANN.—A. f. Psych. u. Nervenhk., xx, 1888;

Temporal side.
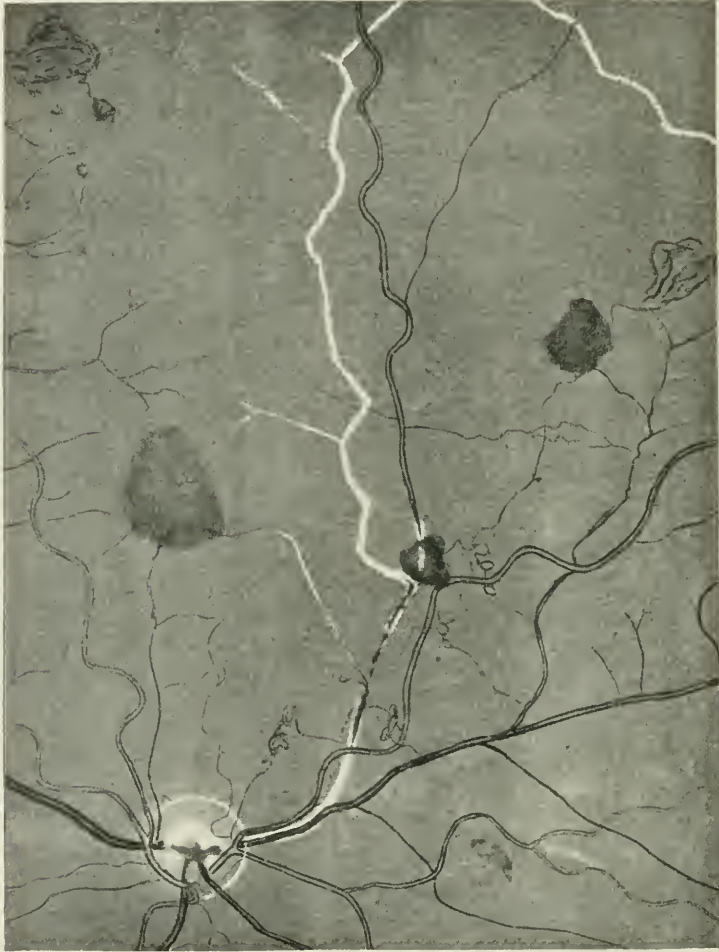
Nasal side.

FIG. 831.—VASCULAR DEGENERATION.

Inglis Pollock, T. O. S., xxvii. Showing degeneration of vein, telangiectases, and vessels of new formation, in a patient with right hemiplegia.

Z. f. klin. Med., xvi, 1889; K. M. f. A., xxvii, 1889; Z. f. A., vii, 1902. THOMA.—A. f. O., xxxv, 2, 1889. FRIEDENWALD.—Jl. Am. Med. Assoc., 1891; A. of O., xxv, 1896. GARNIER.—C. f. A., xvi, 1892. *MARCUS GUNN.—T. O. S., xii, 1892; xviii, 1898. HERTEL.—A f. O., lii, 1900. *DE SCHWEINITZ.—Path. Soc., Philadelphia, 1897–99; Maryland Med Jl., 1900; T. Am. O. S , 1906; Internat. Clinics, i, 1907. ALLEMAN.—Am.

Med., 1904. Woodruff.—Tr. Am. Acad. of O., 1905. Rohmer.—Soc. franç. d'O., 1906.
Coats.—Ophthalmoscope, iv, 1906. Pollock.—T. O. S., xxvii, 1907. Siegrist.—Internat.
Ophth. Congress, Utrecht, 1899. Zimmermann.—A. f. A., xli, 1900. Sous.—Ann. d'Oc.,
liii, 1865. Magnus.—Virchow's Archiv, lx, 1874. Litten.—Berliner klin. Woch., 1881.
Fuchs.—A. f. A., xi, 1882. Uhthoff.—B. d. o. G., 1883. Benson.—T. O. S., ii, 1882.
Schleich.—Mitt. a. d. ophth. Klinik in Tübingen, 1885. Story.—T. O. S., vi, 1886.
Schmall.—A. f. O., xxxiv, 1, 1888. Seydel.—A. f. A., xxxviii, 1889.

Thrombosis of the central vein of the retina manifests itself clinically
as an extreme form of "hæmorrhagic retinitis," almost always unilateral,
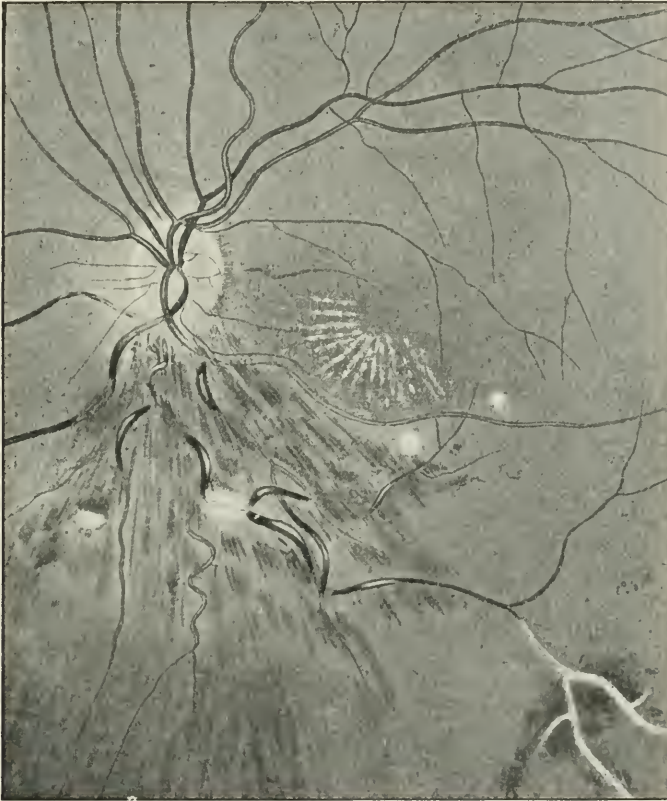
Fig. 832.—Thrombosis of Retinal Vein.
Parsons, Diseases of the Eye. Thrombosis of a branch of the central retinal
vein. From a case of W. T. Lister.

of sudden onset, involving the whole area drained by the central vein
or the area of only one branch. The pathological aspects of the
disease have been admirably investigated by Harms and Coats. The
following is for the most part derived, often verbatim, from Coats's
paper.

Arguing merely from the clinical standpoint, when one sees the
large hæmorrhages and the enormously swollen and tortuous veins, at
one place buried in exudation, at another thrusting forth a knuckle,

one is driven to the assumption that there must be something obstruct-
ing the venous return; and when one further finds that these appear-
ances are usually confined to one eye, the other fundus being either
normal or showing only slight vascular disease, while there is no
proptosis or other evidence of obstruction of the ophthalmic vein, the
conclusion seems inevitable that the obstruction must be in the central
vein of that eye and not anywhere farther back. These cases occur
usually in elderly people; there are often signs of widespread vascular
disease in the form of thickened peripheral vessels and cardiac hyper-
trophy, and in a certain proportion of instances there is evidence of
nephritis. Hence it seems probable that the obstruction in the central
vein will be a thickening of the vein wall, or a thrombus within its
lumen, or a combination of these two conditions. When it is found
that loss of vision is usually sudden, or at least not very gradual, and
that there are usually not any defi-
nite prodromal symptoms, it might
be concluded that the occurrence
of a thrombosis and not of a very
gradually built-up thickening of the
vein wall would most probably be
the immediate cause of the loss of
vision. Sclerosis of the vein wall
would of course predispose to such
a process of thrombosis.

Fig. 833.—Thrombosis of Central
Vein. × 100.
Coats, R. L. O. H. Rep., xvi, Case 2.
Thrombus in retinal vein; hæmorrhage
into retina.

A large number of cases have
now been examined anatomically.
Coats collected and tabulated 22
(v. Michel [3], Deutschmann [2],
Wagenmann [3], Weinbaum,
Purtscher, Stölting, Goh, Alt,
Fridenberg, Bankwitz, Türck,
Würdemann, Reimar, Gauthier,
Meyerhof, Ischreyt, Yamaguchi),
and added 16. Others have been
reported by Harms (10), Sidler-
Huguenin, Baquis, Bartels, and Verhoeff (6). Cases by Angelucci,
Juler, and Schnabel are inconclusive.

v. Michel seems to be not quite sure whether his own first case
may not have been an obliteration of the vein by proliferation of the
intima rather than a true thrombosis; the picture given of his second
case rather suggests a leukæmic blood-clot with abundant leucocytes,
while the picture of his third case also resembles a coagulum deposited
from stagnant blood rather than a true *intra vitam* thrombus, *i.e.* it
shows neither the lamination of a recent thrombus nor the organisation
of an old one. Similar appearances are not uncommon in the ciliary
vessels and are noted in Coats's Case 3. The same remarks apply to
Gauthier's case, and to Fridenburg's, and probably also to Würdemann's
in which a "clot" is spoken of in both the artery and vein. In
Meyerhof's case it is also doubtful whether there were true thrombi

or coagula.   Bankwitz's explanation of an aneurysm on the artery obstructing the vein is improbable, while Goh's case belongs to a somewhat different category from those under consideration, since it occurred during an exhausting illness and only a few days before death.

Shortly to summarise the different pathological changes which have been put forward in explanation of the clinical picture of "thrombosis of the central vein" we have : (1) a thrombus in the central vein in eleven out of twenty-two cases. (2) Occlusion of the central vein by proliferation of the intima, but without thrombosis (possibly in v. Michel's first case).   (3) Multiple thrombi in the retinal veins, but without a thrombus in the central vein (Alt, Ischreyt). (4) Multiple emboli, or perhaps thrombi in the retinal arteries (Wagenmann, Case 1).   Stölting seems to regard his case as of the same sort.   In Fridenberg's and Meyerhof's cases there were thrombi (or coagula) in both the arteries and veins. (5) Changes in the retinal vessels (hyaline thickening, endarteritis, endophlebitis), sometimes amounting to occlusion, but not the result of either thrombosis or embolism (Wagenmann, Case 3, Deutschmann, Case 2, Reimar) : (6) Hæmorrhage into the substance of the optic nerve (Deutschmann, Case 1).

No remarks are needed on the first three of these explanations, since it is sufficiently obvious how the ophthalmoscopic appearances might be produced by them. With regard to (5) also there is no insuperable difficulty in accounting
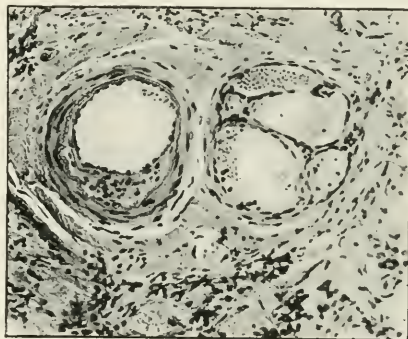
FIG. 834.—THROMBOSIS OF CENTRAL VEIN. × 120.

From the same specimen : central vessels. Artery, to the left, shows endarteritis. The lumen of the vein is divided into loculi containing blood.

for the clinical picture if we suppose the thickening to fall chiefly on the vein walls, and to produce obstruction of the venous current. It is not so plain, however, why lesions on the arterial side of the circulation, whether emboli, thrombi, or endarteritis, should give rise to the appearances of venous obstruction and to the copious hæmorrhages.   The explanation usually given is that there are multiple hæmorrhagic infarcts such as occur when the pulmonary or any end artery is blocked, i. e. that when an obstruction occurs in an artery to which there are no sufficient collaterals, and when in consequence the blood-flow through the corresponding vein is stopped, there occurs a back flow of venous blood in a direction the reverse of the usual, which causes engorgement of the vein and hæmorrhages from it.   A case of Knapp's is usually quoted in which the ophthalmoscopic appearances seemed to indicate that there had occurred an embolism of a single arterial branch in the retina, and in which the corresponding vein was engorged and accompanied by

hæmorrhages. Haab, however, has suggested that the real explanation of this case was that a thrombus in the vein had been the primary lesion, and that in consequence of the obstruction to the blood-flow an endarteritis had been set up in the artery, which gave it the thin appearance of an artery in which an embolus had lodged. It has been abundantly proved, and is indeed a matter of common experience, that branch embolism, like embolism of the main trunk, is not usually followed by any such hæmorrhages as those described in Knapp's case. Haab quotes twenty authorities in support of this statement, and in three cases of branch embolism personally observed Coats never saw any trace of hæmorrhage. It must therefore be considered doubtful whether obstructions on the arterial side of the circulation offer any explanation of the ophthalmoscopic appearances in these cases.
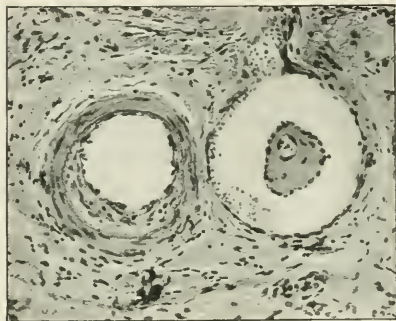
FIG. 835.—THROMBOSIS OF CENTRAL VEIN.
× 120.

From the same specimen, posterior to Fig. 834. The organised tissue in the centre of the vein is a process of the thrombus. It is covered with endothelium, and has a small lumen in its centre lined by endothelium and containing blood. A small branch enters the vein at this point.

Deutschmann's finding of hæmorrhages in the optic nerve as a cause of "thrombosis of the central vein" has apparently never been repeated. The frequency of the occurrence of glaucoma in the cases tabulated may be merely noticed at present.

Certain questions arise on studying these results. Is the clinical picture known as "thrombosis of the central vein" capable of being produced by more than one kind of lesion, as is suggested by the variety of the findings in the above list? If so, is there any means of distinguishing clinically what lesion is present in any given case? If not, if there is some constant pathological entity as the basis of the well-defined clinical entity, how can the variety in the pathological explanations be reconciled? These questions cannot yet be answered with any degree of certainty, and though it is now twenty-six years since v. Michel published the first case, there is still need for the collection of fresh material. Several cases are recorded in which on pathological examination no thrombus or other impediment was found in the veins. It is possible that in some of these cases there really was an obstruction to the vein which was overlooked in the pathological examination. Such a possibility exists, of course, where the whole length of the vein has not been examined in serial section, and in order that no part may be overlooked it is always desirable that the optic nerve entrance should also be cut in *transverse* serial section, and not horizontally in the usual manner. A study of the literature will show that the favourite seat for the lodgment of thrombi is either at the lamina cribrosa or a short distance behind it, probably because the central

vessels narrow as they go through (Hertel). The place where the central vein makes its exit from the nerve is also stated by v. Michel to be a favourite seat, from the obliquity of the course of the vessel in this position, but a case of his own and one of Yamaguchi's seem to be the only ones on record of thrombus in this position. It may therefore easily happen that a thrombus may be overlooked, on the one hand at the lamina because the sections have been longitudinal to the nerve, and on the other hand at the exit of the vein from the nerve if this point be not included in the piece of nerve excised.

There is a further difficulty in the interpretation of the pathological appearances in some of these cases. In many of them the initial lesion occurred long before the eye came to excision, and during part of that time glaucoma was present. It becomes difficult to say, therefore, what changes were primary and what secondary, for it is likely that in an eye long the subject of glaucoma, interference with the intra-ocular circulation will itself give rise to vascular changes and even to thrombosis in the retinal veins, and though it is improbable that increased intra-ocular tension should give rise to a thrombosis in the central vein behind the lamina, yet even this is possible as a result of slowing of the intra-ocular circulation. Then, again, it does not follow in every case of glaucoma in which on bisecting the eye retinal hæmorrhages are found, that these hæmorrhages have preceded the onset of glaucoma as is the usual history in true cases of thrombosis of the central vein. It is necessary, therefore, in each case, unless it is known from actual observation that the retinal hæmorrhages preceded the glaucoma, to weigh the clinical with the pathological evidence before coming to a conclusion with regard to the sequence of events.

FIG. 836.—THROMBOSIS OF CENTRAL VEIN. × 120.

Coats, R. L. O. H. Rep., xvi, Case 5. Showing the central vessels in the anterior part of the lamina cribrosa. The vein, above, is occluded by a homogeneous mass containing a few leucocytes and other cells. In the artery the elastic membrane has separated from the media, probably an artefact.

If a history be given of sudden loss of vision in the form of clouds and mist before the eyes, perhaps noticed on waking in the morning, if there have been at first no pain or redness of the eyeball, but if these symptoms have made their appearance later, then the probability is strong that the case was indeed first one of thrombosis of the central vein upon which glaucoma ensued. If, on the other hand, the history be of loss of vision with supra-orbital neuralgia, pericorneal redness and rainbows round lights, then it is probable that the glaucoma was the first fact and that the hæmorrhages ensued later, either as a consequence of vascular disease or as a result of operative interference.

The following, then, are desirable conditions in the material upon which we conduct our investigations on thrombosis of the central vein.

The disease should not have lasted too long, otherwise it will be difficult to separate primary from secondary lesions. Glaucoma should not have been present, since it complicates matters, or if it has been present (for we seldom obtain pathological material in which it has not occurred), it should not have lasted long enough to mask the earlier condition of affairs. It should be known by actual observation, or rendered probable by the history of the case, that the hæmorrhagic retinitis and not the glaucoma was the first factor in the case. The eye should not have been too much interfered with by operation. The eye should be cut in serial section, and more especially the disc and lamina cribosa should be cut in transverse serial section.

Coats has recorded four cases in which the appearances known clinically as "thrombosis of the central vein" were either seen ophthalmoscopically, or were found macroscopically when the eyes were bisected after hardening. Of these, three showed evidence that there had really been a thrombus to account for the symptoms, and the successive stages in the processes of thrombosis and organisation may be roughly indicated by Figs. 833 and 834. Some uncertainty remains with regard to Coats's Case 1, because the optic nerve entrance was cut longitudinally, for it will be readily understood how little could have been said with certainty of the changes shown in Figs. 836 and 837, for instance, if the sections had passed longitudinally to the nerve entrance in the usual manner instead of transversely.

FIG. 837.—THROMBOSIS OF CENTRAL VEIN. × 120.

From the same specimen, farther back. The vein, A, is obliterated. Note the density of the connective tissue around the central vessels in the lamina —a normal feature.

The chief problems which require solution are: (1) What general and local conditions favour the occurrence of thrombosis of the central vein? (2) Why does glaucoma so commonly follow in these cases? (3) What is the exact nature of the changes found in the central and retinal vessels, and how much of them is primary and how much secondary to the onset of thrombosis and glaucoma?

Chief among the causes of thrombosis of the central vein must undoubtedly be counted angio-sclerosis, including in this term all those conditions which tend to the thickening of vessel walls and the narrowing of their lumina, and including affections both of arteries and of veins. An examination of the literature shows that out of 26 cases arterio-sclerosis of the systemic vessels was present in 11, absent in 8, and not mentioned in 7. Schoenewald found arterio-sclerosis present in 7 out of 18 cases. In 9 cases observed by Coats it was present in 4 and absent in 5. These figures do not give so large a proportion with arterio-sclerosis as might have been expected from some of the descriptions of the disease, in which rigidity of the radial and temporal

arteries is stated to be nearly always present. It may be noticed in passing that while arterio-sclerosis of the peripheral vessels is a valuable guide pointing to the probability of arterio-sclerosis being present in the vessels of the eye, its absence cannot be taken to mean absence of vessel change within the eyeball.   In Coats's Case 1, for instance, in which the changes in the retinal vessels reached the very high degree shown in Fig. 436, there was no evidence of arterio-sclerosis in the peripheral vessels.   The same is well known to obtain in the case of the cerebral vessels, which are sometimes highly sclerosed, although no evidence of general arterio-sclerosis is furnished by the peripheral arteries.   There may even be extensive microscopic changes in the retinal arteries without ophthalmoscopic evidence as Hertel has recently shown.   It is, however, true that in nearly all the cases of thrombosis of the central vein which have come to microscopic examination the vessels have been found extensively diseased.

It follows that if angio-sclerosis be a cause of thrombosis, the conditions which favour the occurrence of angio-sclerosis must also be included among the causes of thrombosis.   Of these the chief are old age, heavy strains, nephritis, and perhaps syphilis.

The influence of old age is best represented in tabular form, as follows :—

Age.			Cases from literature.		Coats.
1—20	.	.	1	.	—
20—40	.	.	5	.	2
40—60	.	.	8	.	6
60—80	.	.	10	.	1
80—90	.	.	1	.	—

FIG. 838.—THROMBOSIS OF CENTRAL VEIN. × 120.

From the same specimen, behind the lamina.

The figures of Ammann may also be quoted, who found among 60,000 patients in Haab's clinique 20 cases of thrombosis of the main venous stem, of whom 15 were between the ages of 50 and 80, and 2 of the others, æt. 47 and 29 respectively, had heart disease and arterio-sclerosis.   Their figures are sufficient to show how the disease increases with increasing age—a fact which is of common experience. Nearly all the cases occurring under 40 are of an unusual type, and in some of them the diagnosis is doubtful.

Ammann has especially called attention to the frequency with which those suffering from thrombosis of the central vein are persons accustomed to much toil, and especially to much stooping.   He found that 13 out of his 20 cases were either peasants or persons who did gardening for pleasure, while 2 others were smiths.   We have here perhaps both a remote and an immediate cause of the affection, since constantly repeated strains would tend to cause angio-sclerosis, while the venous congestion caused by some specially violent strain might be the cause of the actual thrombosis.   It is because of their greater

liability to physical strains Ammann believes that more cases occur among men than among women.

With regard to the influence of nephritis, it will be found that this is less prominent than might have been expected. Out of 34 recorded cases it is only mentioned 13 times, in 7 it is not noted whether it was present or absent, and in 18 it was not present. In any case it is to be regarded not as a direct, but as an indirect cause; it will act presumably merely by helping in the causation of angio-sclerosis.
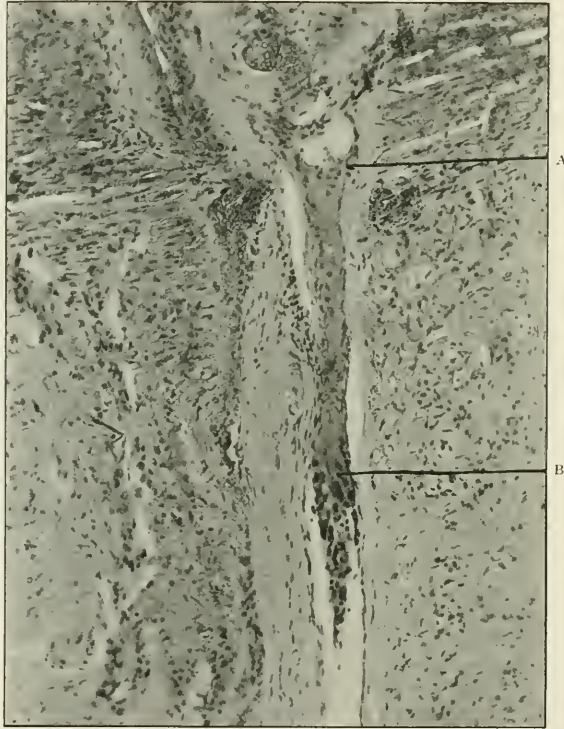
Fig. 839.—Thrombosis of Central Vein. × 120.

Coats, R. L. O. H. Rep., xvi, Case 11. Longitudinal section of nerve. From the region of the lamina cribrosa, A, backwards, the vein is occupied by a homo-geneous coagulum. There is slight endothelial proliferation. At B the coagulum is invaded by cells, polymorphonuclear leucocytes, and larger paler cells.

Syphilis is usually credited with having a considerable influence in the ætiology of angio-sclerosis, e.g. the frequent history which one obtains of it in cases of aneurysm. Yet it is very seldom mentioned in the reports of cases of thrombosis of the central vein.

Angio-sclerosis, then, may be given the first place in causing throm-bosis of the central vein. Yet all cases do not seem to be so explained, and among other causes valvular heart disease must be mentioned.

Coats records a case in a young woman with mitral stenosis. A somewhat similar case of Juler's may be mentioned. Valvular disease

has also been present in some of the other cases in the literature (*vide* v. Michel's Case 1, Türck, Meyerhof), but either associated with evident arterio-sclerosis, or occurring at an age when arterio-sclerosis might be expected to be present. Schoenewald gives three cases out of his eighteen as suffering from heart disease.

It might be expected that weakening illnesses might occasionally be followed by a thrombosis of a marantic nature, but it seems that this is seldom the case. The patients are usually apparently in their ordinary health. Coats has in three cases obtained a history of influenza, a disease which is known sometimes to cause thrombosis in the cerebral vessels. In two dimness of vision occurred during convalescence, and in one about a month after an attack. To a similar category may be referred the case of Würdemann, in which thrombosis of the central vein followed a fortnight after an attack of mumps in a boy of eight. Also possibly marantic was Case 2 of v. Michel, in which a thrombus was found in the central vein of a patient who died of leukæmia, and a case of Ballaban's (not proved pathologically) in a girl suffering chlorosis. Goh's case may also be mentioned here, in which a hæmorrhagic retinitis appeared three days before death from septicæmia, and in which a thrombus was found in the right central vein. It is a noteworthy point (first mentioned by v. Michel) that in not a few cases dimness of vision has been first noticed on awakening in the morning. This was the history in the cases of Bankwitz and Wagenmann (Case 1), and Coats obtained it in three out of nine cases. It probably merely signifies that, given conditions favourable to the occurrence of thrombosis (angio-sclerosis, etc.), the lowered blood-pressure and slower current during the hours of sleep are likely to complete the process.

Among strictly local conditions favourable to, but not necessarily by themselves causing thrombosis of the central vein, may be mentioned an unusual course of the vein itself, since it is evident that the more curved or tortuous the tube is along which the blood flows, the greater will be the friction, the greater the slowing, and the more the liability to thrombosis if the wall be diseased. There is a certain amount of proof that these considerations are of some little importance. Goh believed that in his case a thrombosis occurred in one eye and not in the other because the vein on that side took an unusually great curvature, and in Coats's Case 2 the vein just central to the thrombosed part took an unusual course, curving sharply away from the artery to reach the periphery of the nerve.

When all the above causes have had due weight, there still remain certain cases not easily explained. These are the cases occurring in young people without cardial or renal disease. The cases of Purtscher and Weinbaum may be especially mentioned, which occurred in patients æt. 21 and 25 respectively, and without heart disease, arterio-sclerosis, or nephritis. Coats has also seen a similar case in a young lady, which was further remarkable in that the hæmorrhage subsequently entirely disappeared and the vision rose to $\frac{6}{6}$. I have recorded a similar case. These cases are not easy to account for since thrombosis in any vein is uncommon in the young, except in the two

instances of varicose veins and the uterine veins after parturition, in both of which instances there are special circumstances not to be found in the central vein of the retina.

Several explanations are put forward of glaucoma following these cases of intense hæmorrhagic retinitis. That glaucoma does follow oftener than can be explained on the ground of mere coincidence is a well-ascertained fact. So long ago as 1869, before thrombosis of the central vein had been described as such, v. Graefe gave his experiences on this point and stated that from the number of cases in which he had seen this sequence of events he could not regard the occurrence of glaucoma as accidental. One explanation which has been put forward, chiefly by Wagenmann, is that the two phenomena, hæmorrhagic retinitis and glaucoma, are really independent of one another, but are both dependent on a common cause, viz. arterio-sclerosis, i.e., that the vascular changes which in the posterior half of the eye resulted in hæmorrhages, in the anterior half of the eye resulted in impaired drainage and glaucoma. A little consideration of this theory will show that it is untenable. If the glaucoma is not directly dependent in some way on the hæmorrhagic retinitis, why should it attack the eye affected with hæmorrhagic retinitis rather than the other as it undoubtedly does? v. Graefe observed twenty-two cases in which glaucoma followed upon hæmorrhagic retinitis. In ten of these the other eye was unaffected after a lapse of two years. In six glaucoma followed in the other eye, but was in each case preceded by retinal hæmorrhages. In five the other eye was subsequently affected with hæmorrhagic retinitis but remained free from glaucoma, and in one both eyes were simultaneously attacked. Presumably on an average the amount of angio-sclerosis in one eye will be much the same as in the other eye of the same person, and if the glaucoma is not dependent on the retinal hæmorrhages, but on angio-sclerosis, there seems to be no reason why it should fall with greater frequency on the eye affected with retinal hæmorrhages than on the one not so affected, but the above figures show that there is no tendency for the other eye to become glaucomatous, except in consequence of the occurrence of retinal hæmorrhage in it. Again, the hypothesis that both are due to the common cause, angio-sclerosis, does not explain cases such as those of Weinbaum, Purtscher and Würdemann, in which hæmorrhagic retinitis followed by glaucoma occurred in young patients, since in these cases the glaucoma cannot have been due to angio-sclerosis, and must undoubtedly be looked upon as a sequel to the retinal hæmorrhages. It may be concluded that the two conditions are not independent, but that the glaucoma is directly the consequence of the retinal affection.

In a few cases there has been evidence of iritis (Wagenmann, Case 3, Gauthier, etc.), and where this has occurred there would, of course, be an increased tendency to glaucoma, but that this is any usual explanation of these cases is easily negatived by most of the clinical histories. It is distinctly uncommon to find any trace of inflammation in the stroma of the iris microscopically.

There are also certain instances in which glaucoma has followed

upon the use of atropin (Deutschmann), but this also is quite an exceptional history in these cases, and probably indicates that the eye was already predisposed to become glaucomatous.

It has been supposed that in these cases there has always been a rupture of the blood through the limitans interna into the vitreous. In consequence of this, it is thought, the lens may be carried forward, bearing with it the iris and narrowing the corneo-iridic angle. At the same time the nature of the fluid drained off at this angle will be altered ; it will become more colloidal from admixture with the proteid constituents of the blood, and will, therefore, be less easily able to pass through the spaces of Fontana, and these two factors combined will result in increased intra-ocular tension. Some support is lent to this explanation by the fact that in many instances, with the onset of glaucoma the media become too opaque to be illuminated, and it is also undoubtedly true that blood has actually been found in the vitreous in some of these cases. No doubt it is the case that the presence of blood in the vitreous will increase the tendency to glaucoma, but it is quite certain that glaucoma may also occur without any trace of such vitreous hæmorrhage.

It is, however, possible that without actual hæmorrhage into the vitreous the lymph currents drained at the corneo-iridic angle may still be altered in the manner indicated by the presence of profuse retinal hæmorrhages and retinal venous obstruction. It is to be remembered that the canal of Stilling is capable of being injected from the lymph spaces around the central vessels (Leber,) which shows that venous (and therefore lymphatic) obstruction in the nerve may alter the constitution of the lymph currents in the vitreous.

Such an alteration in the lymph currents may be the explanation of the onset of glaucoma in these cases, but is not the full explanation of its continuance. We have not merely to do with the drainage of abnormal lymph through normal passages, for already on the seventh day, as in Coats's Case 1, there is a certain amount of blockage of the corneo-iridic angle by fibrous tissue—the apposition has become an adhesion—and this is a condition which constantly increases the longer the glaucoma persists. It is noteworthy that the corneo-iridic angle is often much more occluded on the temporal than on the nasal side (Coats). It is to be remembered also that glaucoma does not follow in every case of thrombosis of the central vein. Possibly the imperfectly understood factors which predispose to ordinary primary glaucoma play a part, e. g. general smallness of the eye, shallowness of the anterior chamber, relative largeness of the lens, and angio-sclerosis. It is to be remarked, however, that in Coats's Case 1 the anterior chamber was not shallow but rather deep. Angio-sclerosis is probably an important factor in these cases, not as an independent cause of glaucoma, according to Wagenmann's explanation, but as the predisposing cause which makes the drainage fail when the abnormally constituted lymph attempts to pass through the filtration angle.

Finally, what is the nature of the changes in the vessels described and figured in the foregoing account ? Are they all of one kind, the different appearances being different stages in one process, or is there

more than one kind of pathological change which the central vessels may suffer ? Further, are the changes in the arteries of the same nature as the changes in the veins ? In the first place, a few words may be said as to the peculiarities in structure of the central vessels and their retinal branches, and more particularly as to the elastic tissue in their walls, since the staining of this elastic tissue by special methods, and especially by Weigert's elastic tissue stain, has given much information recently both with regard to the normal structure of the vessel walls and with regard to the seat of pathological changes in them. It has also been of much service in distinguishing in the case of the retinal vessels which are arteries and which veins—a distinction by no means easily made when they are pathologically altered. The central artery, then, as far as the lamina, has the normal structure of a small arterial twig—an endothelial lining, a very narrow sub-endothelial layer, a well-marked crenated elastica interna, a relatively thick muscular coat with scanty elastic fibres, and a connective-tissue adventitia fading indefinitely into the surrounding connective tissues, and containing an abundance of irregularly disposed fine elastic fibres. So far there is nothing peculiar, but just as the artery passes through the lamina cribrosa there is a change. There ceases to be any definite single crenated membrane, and its place is taken by an entanglement of fine elastic fibres, which still separates media from endothelium. Following the arteries further into the periphery of the retina, this elastic tissue soon becomes less, and in the smallest twigs seems to disappear. The wall of the central vein is composed almost entirely of fine connective tissue, only a very few cells being present which resemble muscle cells. This fibrous tissue has no clear outer boundary, but shades off into the surrounding connective tissue quite indefinitely. It contains tolerably abundant irregularly disposed elastic fibres, which are very variable in amount, and apparently increase, as Hertel has shown, with advancing age. Sometimes these are so abundant, and so regularly arranged in a transverse direction, as to simulate a crenated membrane, more especially when the vein is collapsed and thrown into folds, but the existence of a true membrane in the vein like the crenated membrane of the artery cannot be demonstrated ; the high power always shows that we are really dealing with a close feltwork of fine fibres, an appearance which is not given with any power by the true crenated membrane of the artery. The distinction between intima and media is therefore much less distinct in the vein than in the artery, indeed, the intima seems to consist of little more than the endothelium, with perhaps a little sub-endothelial tissue. On reaching the lamina cribrosa the elastic tissue of the vein wall blends with the elastic tissue, of which the lamina is mainly composed ; and within the lamina there is no elastic tissue in the vein wall, and we have, therefore, in Weigert's elastic tissue stain a valuable means of distinguishing artery from vein, when the ordinary means—the wider calibre, extreme thinness, and lack of muscular tissue in the wall—fail in consequence of pathological changes. It is well known, of course, that there is no muscular tissue in the walls of the retinal veins.

In Coats's cases there is not much difficulty in defining the seat of the pathological change in the arteries. A glance at Fig. 839 will show even without Weigert's special stain that the intima is the part affected, and since the lumen is still lined with a tolerably even layer of endothelium it is probable that the seat of change is the sub-endothelial tissues, though it is not entirely excluded that the endothelium itself may have become proliferated while still presenting an even layer to the lumen. Weigert's elastic stain shows this even more clearly by defining the elastica interna, and further shows that among the proliferated cells there are numerous very fine elastic fibres which form imperfect crenated membranes within the true one, reaching each a part of the way round the lumen, but none entirely round. Within the lamina cribrosa the changes in the arteries are similar. They affect the intima, and seem to consist chiefly of proliferation of the endothelium.

In Case 1 the changes in the central vein are also undoubtedly in the intima. They are in the form of a clear-looking ground worked with scanty oval nuclei, and are within the area of the curly elastic fibres of the media and adventitia. Changes such as are shown in Case 3, however, seem to involve also the connective-tissue wall of the vein. The thickening is very great, there is a certain faint fibrillation in the groundwork, and Weigert's elastic tissue stain shows a very few faint elastic fibres. The endothelium shows but slight sign of proliferation. The changes in the retinal veins are probably usually of this kind.

The terms "hyaline thickening" and "hyaline degeneration" have apparently been used somewhat loosely by authors to represent any thickening of the retinal vessel walls of more or less homogeneous aspect. They should be used only for those cases in which there is evidence of molecular impaired vitality in the tissues, in the form of loss of fibrillar structure in connective tissues, and of lost or much impaired nuclear staining. On this standard none of the changes just spoken of should be termed "hyaline degeneration," for in spite of the somewhat homogeneous ground substance in some instances the nuclear staining is well preserved. "Fibrosis" would be a more appropriate term for some of the changes, and they are no doubt to be looked upon as a preservative tissue reaction against abnormal circumstances in the circulation, a strengthening of the vessel wall against undue strains. Some of the vessels in these sections do, however, show a change different from the above. They are mostly small vessels, have a homogeneous glistening aspect and show no, or very scanty, nuclear staining. To these the term "hyaline degeneration" may be fairly applied without committing ourselves to any statement as to the exact nature of the change, and as to whether it was a primary affection of the vessel wall or followed on the fibrosis above mentioned. An example of what may be regarded as true hyaline degeneration in the retinal vessel walls will be found described and figured by Spicer and Parsons (v. Vol. II, p. 588). What the chemical nature of this change is seems to be quite uncertain; indeed, the different names applied to it—"hyaline," "colloid," "amyloid," "mucoid"—might

lead one to suspect as much.   It stains pink with v. Gieson's fluid.

It seems probable, then, that more than one type of change may be present in the retinal vessels.   We may have the intima taking the chief part as in the arteries in Case 1, and in the vein far back in the nerve in the same case.   To this the terms "endarteritis" or "endophlebitis" may be fairly applied if we keep clearly in mind that there is no evidence of inflammation in the ordinary sense (round-cell infiltrations or cicatrisation) but merely of proliferation.   Then, again, we may have the connective tissue of the wall playing the chief *rôle* as, for instance, in the vein in Case 3, central to the obliterated portion.   To this the terms "arteritis," "periarteritis," "phlebitis," and "periphlebitis" may be applied, again keeping in mind that there is seldom much proof of inflammation.   We may also, of course, have combinations of these two conditions as in the retinal veins in Case 1. In addition to these forms we may have, whether as a primary disease of the vessel wall or as a consequence of such changes as the above, the condition known as "hyaline degeneration."   We may also have the appearances produced by the organisation of thrombi within the vessel lumen, but this, of course, is not a primary disease of the vessel wall.

It is likely that such thickenings of the vessel wall do not lead to hæmorrhage at the actual point of thickening.   The wall will be rather strengthened at this point, for there is no reason to think that the new-formed substance is any weaker than the original wall.   The hæmorrhages are no doubt produced by the damming back of the blood owing to the narrowing of the lumen, and by the giving way either of unthickened veins more peripherally, or of capillaries which are incapable of much thickening.

The question has been more than once raised how much of the condition of the vessels described above was primary, *i.e.* was present before any hæmorrhages or dimness of vision had occurred, and how much was secondary to impeded venous flow from thrombosis and glaucoma, and to the general prejudicial effects of continued high tension on the intra-ocular structures.   Some observations by Hertel are of importance in this connection.   Struck with the difficulty of distinguishing primary from secondary lesions where a hindrance to the ocular circulation has lasted for some time, Hertel examined microscopically seventeen eyes from persons who had general arterio-sclerosis, but no ophthalmoscopic evidence of angio-sclerosis, and also a number of eyes from persons who had neither general arterio-sclerosis nor ophthalmoscopic changes.   He describes two kinds of change. Firstly, an increase of the elastic tissue of all the coats of the vessels, with a tendency to splitting of the elastica interna into more than one layer, but without any definite narrowing of the lumen of the vessel. Secondly, localised bulgings into the lumen consisting chiefly of proliferated endothelial cells with new-formed elastic fibres among them, and proliferation of the sub-endothelial tissues.   The first of these changes he regards as not truly angio-sclerotic, but merely senile, because in the examination of normal eyes of different ages he found a very definite increase in the elastic tissues from childhood to manhood,

and, to a less degree, from manhood to old age.   The second group of changes Hertel regards, however, as really pathological, and as being the changes which precede and produce the retinal hæmorrhages of old people.   An interesting point in his observations is that he found these changes tolerably advanced in eyes which showed no evidence whatever ophthalmoscopically of any vascular disease.   Hertel dealt chiefly with the arteries, but there is no doubt that such changes also occur in veins. Sections from Case 1 show very extensive disease in retinal veins, yet hæmorrhages had only been present for three weeks, and glaucoma for one week.   It is probable, therefore, in this case that there had not been time for the development of any very extensive changes in the retinal vessels, and that the condition shown in these figures approximately represents the condition present while the eye was still an organ of normal function.

Coats's further investigations led him to conclude that obstruction in the vein fully accounted for the symptoms, and that in a very large proportion of cases there was an actual thrombus.   This eliminates two of the six possible theoretical explanations which have been put forward, viz. (1) multiple emboli or thrombi in the retinal arteries, and (2) changes in the retinal vessels—hyaline thickening, endovasculitis— not the result of embolism or thrombosis.   Another explanation, hæmorrhage into the substance of the nerve (Deutschmann), has not been confirmed.   Multiple thrombi in the retinal veins without thrombosis of the central vein is theoretically tenable, but has not been proved. Extensive thrombosis of the retinal veins has, however, been proved associated with thrombosis of the central vein.

Harms has reported two cases of arterial and ten of venous obstruction.   Of the latter, one seemed to be due entirely to obliteration of the vein from endo- and meso-phlebitis, without thrombosis.   One was due to an organised thrombus, apparently associated with a normal vein wall. Three were due to thrombosis associated with disease of the vein wall, the coagulum being laid down at the site of greatest disease in one, somewhat farther up the nerve in the other two.   In one of these two the thrombus was canalised, the case resembling Coats's Case 2, which was the first recorded example of this condition.   In two there was a thrombus in the vein, associated with advanced endarteritis in the artery ; in one of these there was also a thrombus in the artery. In three cases no obstruction was found in the central vein, but in two of these the nerve entrance was cut longitudinally, and in one the nerve was divided very close to the globe, and there was a deep glaucomatous cup, so that almost no part of the central vein was available for examination ; in two some of the retinal veins were thrombosed.   From the consideration of his own cases, and of those recorded in literature, Harms comes to the conclusion that the following lesions have actually been proved by pathological examination in cases clinically diagnosed as thrombosis of the central vein : (1) Thrombosis of the vein ; (2) disease of the vein wall (endo- and meso-phlebitis) ; (3) a combination of these two ; (4) very exceptionally (Yamaguchi) compression of the vein from without.   He is also inclined to admit thrombosis of the retinal veins without obstruction of the central vein.   He regards with

the gravest suspicion attempts to explain such cases by lesions on the arterial side of the circulation. Where the whole course of the vein, and especially that part of it which traverses the lamina cribrosa, has not been examined in transverse sections, there is a strong probability that the essential lesion has been overlooked. The endarteritis may indeed be the cause of the ophthalmoscopic appearances, but not directly, only by slowing the blood current, and so favouring the deposition of a thrombus in the vein ; this again will react prejudicially on the arteries. If such a thrombus is *not* formed in the vein, and if the endarteritis progresses sufficiently, the picture of obstruction of the artery is produced, not that of occlusion of the vein. This also has been definitely proved by pathological examination (Galinowsky, Harms, Coats, and others).

Another example of canalised thrombus has been reported by Sidler-Huguenin. The endothelium of the artery was extensively detached, and lay coiled up in the centre of the lumen—an appearance which the author believed to be due to stripping up by the blood current, such as occurs in dissecting aneurysm. The thrombosis of the vein was supposed to be due to slowing of the blood current from this cause. It is by no means certain, however, that the condition in the artery was not in part an artefact.

Baquis has also reported a case in which an organised thrombus was found in the vein in the region of the lamina cribrosa. In all these cases glaucoma followed the thrombosis at a longer or shorter interval.

Bartels has studied the condition of all the ocular vessels in glaucoma, but without throwing any new light on the connection of that condition with thrombosis of the central vein.

Verhoeff does not admit the frequency of actual thrombosis. He considers that complete obstruction with the classical ophthalmoscopic signs may be produced by endophlebitis proliferans ; the proliferation may involve the subendothelial tissue alone, or the obstruction may be completed by a more active endothelial proliferation within the lumen. He regards the appearance described as a canalised thrombus of the nature of a dissecting aneurysm, the blood from a collateral tearing up the intima of the vessel.

The similarity of the histological appearances depicted by various authors is striking, and the chief differences are to be found in their interpretation.

v. Michel.—A. f. O., xxiv, 2, 1878; Deut. A. f. klin. Med., xxii, 1878; Z. f. A., ii, 1899. Deutschmann.—A. f. O., xxxv, 3, 1879. Wagenmann, Weinbaum.—A. f. O., xxxviii, 3, 1892; B. d. o. G., 1898. Purtscher.—A. f. A., xxxiii, Ergänzungsheft, 1896. Stölting.—A. f. O., xliii, 2, 1897. Goh.—A. f. O., xliii, 1, 1897. Alt.—Am. Jl. of O., 1897. Fridenberg.—A. f. A., xxxiv, 1897. Bankwitz.—A. f. O., xlv, 2, 1898. Türck.—B. z. A., xxiv, 1898. Würdemann.—B. z. A., xxix, 1898. Reimar.—A. f. A., xxxviii, 1899. Gauthier.—Ann. d'Oc., cxix, 1898. Meyerhof.—Z. f. A., iv, 1900. Ischreyt.—A. f. A., xli, 1900. Yamaguchi.—K. M. f. A., xlii, Beilageheft, 1903. *Coats—R. L. O. H. Rep., xvi, 1, 1904; xvi, 4, 1906; T. O. S., xxiv, 1904. *Harms.—A. f. O., lxi, 1905. Sidler-Huguenin.—A. f. A., li, 1904. Baquis.—Festschrift f. Hirschberg, 1905. Bartels.—Z. f. A., xiv, 1905. Verhoeff.—A. of O., xxxvi, 1907. Angelucci.—K. M. f. A., xvi, 1878; xvii, 1879; xviii, 1880. Juler.—Brit. Med. Jl., 1896. Schnabel.—A. f. A., xxiv, 1892. Knapp.—A. f. A., i, 1869. Haab.—In Norris and Oliver, iv, 1900. Schoenewald.—

In Nagel's Jahresbericht, 1900. HERTEL.—A. f. O., lii, 1901. AMMANN.—B. z. A., xxxviii, 1899. BALLABAN.—A. f. A., xli, 1900. EVERSBUSCH.—K. M. f. A., xxxvii, 1899. v. GRAEFE.— A. f. O., xv, 3, 1869. PARSONS.—T. O. S., xxvii, 1907. GALINOWSKY.—A. f. A., xliii, 1901. HUTCHINSON.—Med. Times and Gaz., 1878. AXENFELD.—Berliner klin. Woch., 1896.

DISEASES OF THE ORGANS OF DIGESTION.

Diseases of the mouth may affect the eye by continuity through the circulation or reflexly.   Infective lesions in the mouth may spread by continuity, especially along the veins of the pterygoid plexus, to the orbit, setting up orbital cellulitis (q. v.) or to the cavernous sinus causing thrombosis there (q. v.).

Oral sepsis is an undoubted cause of iridocyclitis, owing to the absorption of toxins into the circulation.   The most serious lesion in this respect is pyorrhœa alveolaris.   It is probable that other forms of subacute or acute endophthalmitis are caused in the same manner (v. p. 1214).

Reflex affections of the eyes from the teeth include amaurosis (Hutchinson, Alexander, Feuer), paresis of accommodation (Schmidt-Rimpler), paresis of the third and seventh nerves (Teirlink, Desmarres, Mengin, Ely, Neuschüler), trigeminal neuralgia, glaucoma (Creniceau, Mooren).   In many of these conditions, particularly glaucoma, the ætiological relationship is by no means certain.

A good example of ocular and dental diseases due to the same cause is found in lamellar cataract (q. v.).

TEIRLINK.—Ann. d'Oc., xix, 1848.   DESMARRES.—Traité, 1858.   HUTCHINSON.— R. L. O. H. Rep., iv, 1865. ZEHENDER.—K. M. f. A., iv, 1866. ALEXANDER.—K. M. f. A., vi, 1868. SCHMIDT-RIMPLER.—A. f. O., xiv, 1, 1868. DIMMER.—Wiener. med. Woch., 1883. POWER.—Med. Times and Gaz., 1883. CRENICEAU.—K. M. f. A., xxiv, 1886. NEUSCHÜLER.—Rec. d'O., 1889. DUNN.—Am. Jl. of O., 1891. COURTAIX.—Maladies des Yeux et Maladies des Dents, Paris, 1892. FEUER.—Klin. Zeit- u. Streit-fragen, x, 1892 (Bibliography). PÉCHIN.—Rec. d'O., 1896. BULL.—Internat. Dental. Jl., 1898. WOLF-BERG.—Woch. f. Ther. u. Hyg. d. Auges, 1898. TH. FUCHS.—Wiener zahnärztl Monats-schrift, 1899 (Bibliography). GROENOUW.—In G.-S., xi, 1, 1900 (Bibliography).

Parotitis is said to cause papillitis (Hatry, Talon, Dor, Blanchard, Woodward), passing on to atrophy, paresis of extrinsic (Dor) and intrinsic (Burnett, Baas) muscles, metastatic iridocyclitis (Schiess-Gemuseus).   Simultaneous affection of the lacrymal gland in mumps has often been described (Rider, v. Schröder, Walter, Gordon Norrie). Woodward found in the literature conjunctivitis (28 cases), optic neuritis and neuroretinitis (23 eyes), dacryo-adenitis (14 cases), iritis (6 eyes), optic atrophy (6 eyes), retrobulbar neuritis (3 eyes), and paralyses (3 cases).   de Micas saw a case of iritis.

Symmetrical inflammation of the lacrymal and salivary glands was described by Mikulicz and confirmed by Kümmel, Tietze, v. Brunn, van Duyse, and others.

RIDER.—Trans. Med. Soc. of New York, 1873. HATRY.—Rec. de Mém. de Méd. milit. 1876. SCHIESS-GEMUSEUS.—Jahresb. d. Augenheilanstalt, Basel, 1882. BAAS.—K. M. f. A., xxiv, 1886. BURNETT.—Am. Jl. of Med. Sc., 1886. ZIEM.—Berliner klin. Woch., 1889. GORDON NORRIE, HIRSCHBERG.—C. f. A., xiv, 1890. FUCHS.—B. z. A., iii, 1891. v. SCHRODER.—K. M. f. A., xxix, 1891. MIKULICZ.—Billroth's Festschrift, 1892. KÜMMEL.—

Mitt. a. d. Grenzgebieten d. Med. u. d. Chir., ii, 1897 (Bibliography). Tietze.—B. z. klin. Chir., xiii, 1897. Walter.—Die ophth. Klinik., 1899. Dor.—Die ophth. Klinik., 1900. v. Brunn.—Beiträge zur klin. Chir., xlv, 1905. van Duyse.—A. d'O., xxv, 1905. Woodward.—Ann. of O., 1907. de Micas.—Rec. d'O., 1907.

Tonsillitis is said to cause conjunctivitis (Ziem), optic neuritis (Menacho), orbital cellulitis (Mitvalsky), paralysis of ocular muscles (not only diphtheritic) (Fütterer, Jacobson).

Ziem.—Allg. med. Centralzeitung, 1886. Menacho.—Internat. Ophth. Congress, Edinburgh, 1894. Mitvalsky.—Contrib. à la Connaissance de la Thrombophlébite, Paris, 1895. Fütterer.—Annals of Ophth. and Otol., 1896. Jacobson.—Beziehungen d. Veränderungen u. Krankheiten des Sehorgans, etc., Leipzig, 1885.

Intestinal disorders may lead to ocular symptoms, such as retinal (Eales) or vitreous (Ziminski), hæmorrhage following straining at stool. Much less authenticated are amblyopia, papillitis, retrobulbar neuritis, etc., due to acute ptomaine poisoning (Galezowski, Clemesha, Young, Santos Fernandez). It is more likely that the continued absorption of ptomaines or bacterial toxins from the intestinal canal may be responsible for iridocyclitis, choroiditis, and other forms of endophthalmitis (Parsons, de Schweinitz).

Galezowski.—Union méd., 1876. Eales.—Birmingham Med. Rev., 1880. Ziminski. —Rec. d'O., 1888. Clemesha.—New York Med. Jl., 1898. Young.—T. Am. Med. Assoc., 1898. Uhthoff.—Internat. Congress, Paris, 1900. *de Schweinitz.—Ann. of O., 1906; T. Am. Med. Assoc., 1907. Parsons.—Brit. Med. Jl., 1907.

Intestinal parasites are responsible for ocular disorders which may be caused reflexly, by absorption of toxic products of their metabolism, by wandering of their embryos, or secondarily through anæmia. Reflex dilatation of the pupils, miosis (Denti), iritis (Furnell), optic neuritis (Meurer), xanthopsia (without use of santonin or picric acid) (Hufeland, Königshöfer), etc., rest on no sure foundation. They have usually been ascribed to the presence of the commoner parasites—*Ascaris lumbricoides*, *Oxyuris vermicularis*, *Tænia solium*, etc. *Cysticercus cellulosæ*, the embryo of *Tænia solium*, occurs in the eye (*v.* Vols. I and II), as also rarely the echinococcus (*v.* Vol. II, p. 438). Œdema of the lids, pain on moving the eyes, mydriasis and paresis of accommodation (Kittel) occur in trichiniasis. Filariasis has already been discussed (*v.* Vols. I and II). *Ankylostomum duodenale* and *Bothriocephalus latus* cause anæmia, often with ocular symptoms. They may set up retinal hæmorrhage (Nieden, Pflüger, Natanson, Reyer, Tschemolossow), nystagmus (Masius and Francotte), etc.

Denti.—Boll. d'Oc., xiii, 1891. Furnell.—Madras Hosp. Rep., 1871. Meurer.— K. M. f. A., xxxii, 1894. Königshöfer.—Die ophth. Klinik., 1898. Rampoldi.—Ann. di Ott., xiii, 1884; xiv, 1885. Molard.—Rec. d'O., 1885. Faravelli.—Ann. di Ott., xvi, 1887. Hogg.—Brit. Med. Jl., 1888. Andogsky.—K. M. f. A., xxxii, 1894 (Bibliography). Varese.—A. di Ott., iii, 1896. Hilbert—Vossius Sammlung, ii, 1897. Kittel.—Allg. Wiener med. Zeitung, 1871. Nieden, Pflüger, Natanson.—Ann. d'Oc., cxviii, 1897. Reyer.—Deutsches A. f. klin. Med., 1886. Tschemolossow.—Die ophth. Klinik, 1904.

Diseases of the liver, especially those associated with jaundice, are liable to be accompanied by ocular symptoms. The yellow coloration of the conjunctiva may persist for years (Taylor). In icterus

neonatorum with purulent ophthalmia the pus is coloured with bile, but tears never participate in the colouration. Xanthelasma (v. Vol. I, p. 9), is said to be commoner in jaundiced patients (Korach, Stiller) ; Hutchinson rarely found jaundice but often disturbance of the liver. Xanthopsia, though described as of constant occurrence in jaundice (Moauro), is relatively rare (Frank 2·5 per cent., Hirschberg, Kohn) ; Hirschberg, Moauro, and Purtscher found the lens and vitreous coloured, in opposition to Moxon. Moauro described transitory myopia of 1·5 D and 2 D in two cases ; it is not due to increase in the refractive index of the aqueous (Hess) (v. Vol. III, p. 929). Retinal hæmorrhages are not uncommon in jaundice (Junge and Stricker, Litten) ; other changes are white spots of fatty degeneration, neuroretinitis (Litten), retinitis (Moauro), especially in phosphorus poisoning with acute yellow atrophy of the liver. Changes in the pigment epithelium have been described in jaundice with night-blindness (Baas, Hori, Purtscher) ; the fundus is tigroid, with small white spots and flecks of pigment ; anatomical investigations are published by Weiss, Baas, Hori (ophthalmia hepatica). Dolganoff noted extreme changes in dogs in which the bile-ducts had been ligatured. Landolt described two cases of retinitis pigmentosa in cirrhosis of the liver. Bamberger and Frerichs first described night-blindness in diseases of the liver. There may be xerosis (Leber, Baas) ; Uhthoff found xerosis and night-blindness in 4—5 per cent. of alcoholic patients, probably due to cirrhosis of the liver. Hori and Weiss describe jaundice, night-blindness, and corneal ulcer ; Purtscher jaundice, night-blindness, xerosis, white spots in the retina, and iritis. Night-blindness has been attributed to the solvent action of bile salts upon the visual purple, and to bacterial activity (Purtscher).

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DISEASES OF THE KIDNEYS.

Albuminuric neuroretinitis. — It was known to Bright (1836) tha visual defects may occur in the course of nephritis and that they are occasionally the first symptom. Türck (1850) and Virchow (1855) first demonstrated anatomical lesions in the retina, and Heymann (1856) and Liebreich (1859) first described the ophthalmoscopic picture. The disease is commonest with the contracted kidney of interstitial nephritis, less common with the large white kidney and the amyloid kidney (Beckmann, Traube, Alexander, Gowers, Robertson, Bull, and others ; it occurs relatively rarely in acute nephritis (c. g., Eyre, Sutton), as after scarlet fever, diphtheria, measles (Horner), lead

poisoning (Pedell), malaria, etc., and in the puerperium. Retinitis has been observed in intermittent albuminuria (Pavy's disease) (Ostwalt). The typical picture may be seen in a pre-albuminuric stage (Vance, Hirschberg, Abadie, Gand, Eyre), probably owing to the scantiness or absence of albumen in interstitial nephritis. The chronic nephritis may be due to disease of the bladder, ureter, or pelvis of the kidney (Nettleship).

It is unnecessary to describe the typical picture in detail here. The moderate papillitis, the white star-shaped figure at the macula, white plaques in the neighbourhood of the posterior pole often forming a snowy white mass around the disc, the diseased retinal vessels and the retinal hæmorrhages are familiar. Extreme swelling of the disc in nephritis without intra-cranial complication is rare in adults, though it occasionally occurs (Gowers, Schmidt-Rimpler), with hæmorrhagic pachymeningitis (Uhthoff), without intra-cranial complication (Kampherstein). I have found that a white star at the macula, associated with choked disc due to intra-cranial pressure, without albuminuria or nephritis, a rare combination in adults (Schmidt and Wegner, etc.), is not very uncommon in children. Schlesinger found hæmorrhages with white spots in 77 per cent., without white spots in 14 per cent. of cases ; there was papillitis alone in 7 per cent. Both eyes are usually affected ; unilateral cases are recorded by Leber, Eales, Yvert, Cheátham, Bull, Moore, Miles Miley, Weeks, Webster, Wehrli. A retina atrophied by previous disease is apparently unable to take on the œdema and other changes of renal retinitis (Nettleship). Cases with a history of successive attacks are recorded by Nettleship. Embolism of the central artery is a rare complication recorded by Schmidt-Rimpler, Plenk, Völckers, Kepincki. Complete blindness from albuminuric retinitis is very rare (v. Graefe, van der Laan, Hirschberg). Complications are vitreous hæmorrhage, hæmorrhagic glaucoma (Wehrli), detachment of the retina (Uhthoff, Eason, v. Michel), generally in the puerperal form (v. Graefe, Brecht, Wadsworth, Lotz, Siler, Scherenberg). Nordenson was able to find only 12 cases of detachment of the retina in this disease, Kunz 5 more ; Schlesinger found 3 cases amongst 43 of retinitis albuminurica, Galezowski 5 in 649 cases. Ewetzky described a case of detachment of the retina and choroid, confirmed anatomically.

As regards the frequency of albuminuric retinitis in cases of nephritis Frerichs found 15 per cent. in 41 cases, Lécorché 22 per cent. in 286 cases, Wagner 6 per cent. in 157 cases, Eales 28 per cent. in 100 cases of chronic nephritis, Miles Miley 31 per cent. in 164 cases of chronic and acute nephritis, Galezowski 31 per cent. in 154 cases, Litten 6 cases in 33 of chronic parenchymatous nephritis. The total works out at 22·4 per cent. in 935 cases.

The age of the patients is usually over 40 years. The earliest recorded age is 5 years (Bull). Miles Miley in 45 cases found 2 under 20, 16 between 40 and 50 ; Schlesinger in 42 cases, one 12½ years old, 11 between 20 and 30, 8 between 30 and 40, 22 over 40. Anderson had a case 9, Lawson and Sutherland one 12 years old. Nettleship's more recent researches tend to show that the condition is somewhat

commoner in children than the above statistics would suggest. In 85 cases, 54 were between 30 and 60, 22 being between 50 and 60. Eales found the largest proportion in the decade 55—65. Nettleship has collected 80 cases of interstitial nephritis in patients under 21 years of age ; in 60 a post-mortem examination was made. Retinitis or multiple retinal hæmorrhages were observed in 31 of these cases, and in 2 out of 3 examples of contracted kidney associated with calculus. The eyes were normal in 10 others; in about 37 no ophthalmoscopic examination is recorded. In 149 cases of parenchymatous nephritis in children definite retinitis was found in 7 ; in 43 the eyes were normal ; in 90 no examination is recorded. In 7 cases of amyloid disease the fundi were normal in one and the others were not examined.

Amongst the total number of 40 cases of retinitis in both groups, the number in which the eye changes were very severe was larger than in any similar consecutive number of cases in older subjects. Both retinæ were detached in 3 patients, very abundant hæmorrhage formed a marked feature in 2 or 3 others, whilst the exudation and swelling were excessive in 3 or 4 more. But we must not hastily conclude from this result that the ocular changes are especially liable to reach an unusually high degree in the young ; for it is likely enough that milder degrees of retinitis causing little interference with sight would have been discovered in a good many more of the children had the ophthalmoscope been used by routine.

A similar remark applies to the apparent scarcity of retinal changes in parenchymatous as compared with interstitial nephritis in the young —the ophthalmoscope was used much less frequently in the parenchymatous cases than in the others.

Amongst 40 cases (interstitial and parenchymatous) Nettleship found 2 cases aged 7 years, 3 of 8 years, 2 of about 9, 3 of 10, 2 of 11, 5 of 12, or in all about 18 below the age of 13 years. Rather more than half of the series (23 cases) were aged from 14 to 21 years. Twenty-six of the whole number were females, 14 males.

Of these 40 young patients with retinitis 24 died at a known interval after the eye condition was first discovered. This interval was $3\frac{1}{2}$ years in 1 case, between 12 and 15 months in 4 cases, between 4 and 7 months in 3 cases, and less than 4 months in 16 cases.

From the data at present available it appears, therefore, that the prognostic importance of the retinal changes in chronic renal disease is certainly not less grave, perhaps more so, in children and young subjects than it has been known to be in adults (v. infra).

As regards sex males are more often affected than females in adults, females than males in the young (Nettleship). Schlesinger in 43 cases found 68 per cent. males and 32 per cent. females ; Miles Miley in 51 cases 38 men (75 per cent.) ; Bull 55 per cent. males and 45 per cent. females. Nettleship in 80—90 cases found 56 males (64 per cent.) and 30 females (36 per cent.). Omitting Bull's cases, in 180 there were 68 per cent. males and 32 per cent. females : this proportion is almost the same as that of the sex incidence of chronic nephritis (Dickenson, West). In patients under 21 years of age chronic nephritis is commoner in females than in males (Handford, Sawyer, Nettleship),

in 80 cases Nettleship found 64 per cent. females and 36 per cent males. On the other hand parenchymatous nephritis in the young is slightly commoner in males than in females (Dickenson, Nettleship). The effect of puberty is not the cause of the excess of females in interstitial nephritis, since the preponderance is less between the ages of 13 and 21 than it is below 13 years. The opinion is gaining ground that syphilis is the commonest cause of interstitial nephritis in children, and the incidence of interstitial keratitis, 63 per cent. females and 37 per cent. males (Hutchinson), is interesting in this connection. Possibly differences of frequency or character in scarlet fever as it affects the two sexes may be a factor. In Nettleship's series hereditary syphilis was proved in only 6 cases of interstitial nephritis, 3 being infants ; it was certainly the cause in 4 parenchymatous cases.

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The albuminuric retinitis of pregnancy forms a distinct class of cases. It has been studied exhaustively by Axenfeld and Silex. The latter observed 35 cases in 7 years. Thompson found 4 cases in 30 of albuminuric retinitis, Völckers 2 in 30 cases, Nettleship 22 in about 100 cases. Silex calculates that the disease occurs once in 3000 pregnancies. It is commonly regarded as a retinitis following acute nephritis, but this conclusion must be accepted with discrimination. The prominent feature of the nephritis of pregnancy is fatty degenera-tion of the tubal epithelium. It is not a true acute inflammation (Herman, Groenouw) and chronic nephritis may supervene with unusual rapidity (Dickenson). It must be borne in mind that ordinary chronic nephritis may have been present before the first pregnancy. The onset of symptoms is relatively prolonged and the retinitis usually appears in the second half of pregnancy, rarely at the time of delivery (v. Graefe, Schmidt-Rimpler). The ophthalmoscopic appearances are

generally the same as in other cases of albuminuric retinitis. It is commonly held that the disease is much more frequent in first pregnancies than in others, but Nettleship's cases do not support this view. In 19 cases the retinitis occurred during the first pregnancy in 4, during the fifth or a subsequent pregnancy in 14. In several cases there was a history of several attacks of pregnancy dropsy, the ages of the patients varying from 29 to 41. In 2 primipara no renal or eye symptoms occurred during subsequent pregnancies (*cf.* Axenfeld, Alt) and the patients lived at least ten years after the attack. Sight may first fail soon after confinement ; some have normal fundi, others traces of past papillitis, none signs of widespread retinitis (Nettleship). In none of the cases was there eclampsia, flooding or obvious kidney disease. The prognosis as regards sight in these cases is probably not so good as is commonly thought, though as regards life it is much better than in other cases (*v. infra*). Silex found return to normal vision in 3 cases out of 35, to $\frac{2}{3}$ in 2 cases observed for four weeks. Only moderate vision can be anticipated if abortion is induced about the seventh or eighth month of pregnancy. Culbertson in 36 cases found normal vision at a subsequent date in 17 per cent., partial in 58 per cent., blindness in 25 per cent. Silex observed complete subsidence of the retinal changes in 2 out of 21 cases. Usually there is partial optic atrophy, white or pigmented spots at the macula, etc. Progressive diminution of vision is not uncommon. It has already been pointed out that detachment of the retina is more frequent in these than in other cases (*v.* p. 1294). Embolism of the central artery (Groenouw), vitreous hæmorrhage (Magnus, Power), etc., are reported. Many cases of the beneficial effect of artificial abortion are reported (*e. g.* Fuerst, Macnamara and Potter, de Lapersonne, Holmes, Risley, Thompson, Wadsworth, Cohn). In a case of Meyer's the retinitis improved but attacks of uræmic amaurosis followed. Ayres and Randolph advise abortion only in the presence of eclampsia.

*Axenfeld.—Monatsschrift f. Geburtshilfe u. Gynäk., 1895. *Silex.—Münchener med. Woch., 1895 ; Berliner klin. Woch., 1895. Thompson.—New York Med. Rec., 1888. W. J. Collins.—T. O. S., viii, 1888. Nettleship.—R. L. O. H. Rep., xi, 1886 ; xv, 4, 1903. Herman.—Allbutt's System, vii, Art. Eclampsia. Groenouw.—In G.-S., xi, 1, 1900 (Bibliography). v. Graefe.—A. f. O., ii, 1, 1855. Schmidt-Rimpler.—Die Erkrankungen etc., Wien, 1905. Alt.—Am. Jl. of O., 1894. Culbertson.—Am. Jl. of O., 1894. Macnamara and Potter.—Lancet, 1878. Emrys-Jones, Salter.—Brit. Med. Jl., 1883. Cohn.— Uterus u. Auge, Wiesbaden, 1890. Randolph.—Bull. Johns Hopkins Hosp., v, 1895. Brecht.—A. f. A., xviii, 1877. Snell.—Brit. Med. Jl., 1895. Cirincione.—Clinica oculistica, 1900. Magnus.—Die Albuminurie in ihren ophth. Erscheinungen, Leipzig, 1873. Power.—T. O. S., viii, 1888. Holmes.—A. f. A., xii, 1883. Risley.—Ophth. Rev., v. 1886. Wadsworth.—T. Am. O. S., 1887. de Lapersonne.—A. d'O., viii, 1888. Meyer.— Z. f. A., ii, 1899. Ayres.—Am. Jl. of O., 1882. Randolph.—Bull. Johns Hopkins Hosp., v, 1895. Ahlström.—Ann. d'Oc., cxxix, 1903. Jacobi.—Jl. of Am. Med. Sc., 1904.

The prognosis as regards life is extremely bad in all cases of albuminuric retinitis except those occurring during pregnancy. Bull in 103 patients found that 87 per cent. died within 2 years : 57 in the first, 18 in the second, 6 in the third, 4 in the fourth, 1 in the sixth year. Miles Miley found that death was much earlier in cases of albuminuric retinitis than in other cases of nephritis : from the date of

first observance of retinal changes most died within 12 months, 2 at 14, one at 18 months out of 164 cases of nephritis (105 with normal fundi, 51 with albuminuric retinitis). Snell in 103 cases found that 57 (67 per cent.) died within the first year, 12 in the second year. Possanner found retinitis albuminurica in 131 (0˙2 per cent.) of 67,000 eye patients. Haab in 39 private patients found that 23 (59 per cent.) died within 2 years, longer periods varied from $2\frac{1}{4}$—6 years in men, $3\frac{1}{2}$—11 years in women; in 33 hospital patients all the men died within 2 years, 68 per cent. of the women. The amount of care and attention that the patients receive is therefore of considerable importance in prolonging life. Trousseau in 45 cases found that 28 (62 per cent.) died within 2 years, 10 lived longer than 2 years, 4 longer than 3 years, 3 longer than 4 years. Haehnle in 98 cases found that 56 per cent. died within the first year, 68˙4 per cent. within 2 years, and 14˙3 per cent. from 2 to 9 years. Nettleship in 42 non-pregnancy cases found that 25 died within 1 year, 9 lived more than 2 years. Detachment of the retina in other than puerperal cases is a bad sign, most of the cases dying quickly (Groenouw).

The prognosis as regards life in the albuminuric retinitis of pregnancy is fair. Nettleship reports that in his 22 cases only 5 deaths are known to have occurred—1 within 2 years, the others $3\frac{1}{4}$, $4\frac{1}{4}$, 7, and 7 years after the retinitis. Seven cases are known to have lived more than 2 years—5, $5\frac{1}{2}$, 9, 10, 11, 13, 24 years.

Bull, Miles Miley, and others.—*See* p. 1298. Snell.—T. O. S., viii, 1888. Possanner.—B. z. A., xv, 1894. Trousseau.—Ann. d'Oc., cxv, 1896. Haehnle.— Dissertation, Tübingen, 1897. Nettleship.—T. O. S., xv, 4, 1903. Greenwood.—Boston Med. and Surg. Jl., 1903. Carpenter.—Ophth. Rec., 1904. Elschnig.—Wiener med. Woch., 1904.

The pathological anatomy of albuminuric retinitis varies with the stage. Throughout its course the evidences of active inflammation are almost entirely absent (Weeks), and the changes are due to passive œdema and the results of degeneration of the vessel walls. Œdema is early a marked feature, the fluid separating the fibres of the nerve-fibre layer, thus causing most swelling where this layer is thickest, viz. at and around the disc. Cystoid spaces occur in the inter-nuclear layer, especially near the macula, filled with clear fluid, hyaline coagula, or fibrinous networks (Fig. 428). Cytoid bodies (*v.* Vol. II, pp. 552, 578) occur in the nerve-fibre layer, though they are not characteristic of albuminuric retinitis, nor are they varicose nerve-fibres as thought by H. Müller, who first described them. The nerve-fibres and ganglion cells are swollen and undergo degenerative changes Hæmorrhages occur principally in the nerve-fibre layer (*cf.* diabetes). Exudates often collect between the retina and choroid, so that localised flat detachments of the retina are produced. They are also found between the retina and vitreous. The exudate often contains proliferated pigment cells, which may cause a grey appearance ophthalmoscopically (Liebreich, Nettleship). The characteristic white spots of the ophthalmoscopic picture vary in nature according to the stage of the disease. That they are certainly sometimes due to fatty degeneration of the

tissues is proved by the use of osmic acid (Leber, Wedl and Bock, Herzog Karl Theodor, and others). On the other hand the greater part of the deposits is not generally fatty in nature. As shown by Nettleship they are not dissolved by ether or chloroform, nor are they removed during the preparation of paraffin or celloidin sections. They consist largely of hyaline deposits, some of which stain deeply with hæmatoxylin (Römer). They may indeed in rare cases become completely calcified, the calcareous material being present in organic combination (Römer).

The anatomical basis of the star-shaped figure at the macula has given rise to much discussion. It is undoubtedly dependent upon peculiarities in the normal anatomical disposition of this region, particularly upon the pronounced differentiation of the radial Henle's fibres here. Dimmer has demonstrated fatty deposits in this layer by osmic acid, and Nuel examined two star figures, observed previously by the ophthalmoscope, and showed the presence of hyaline or fibrillar deposits in Henle's fibre layer. They were absent in the fovea itself.

The changes in the vessels differs in no essentials from those found in other cases of vascular degeneration. They have been specially examined by Herzog Karl Theodor, and many others. Leber described the distension of the veins and capillaries, and the formation of new vessels, which may occasionally be observed ophthalmoscopically. Endovascular obliteration occurs at the same time as localised distension, capillary dilatations being not uncommon. Hæmorrhages are seen anatomically in almost any layer, and changed blood-clots may give rise to amorphous deposits. Similar changes are seen in the choroidal vessels, and hæmor-

FIG. 840.—ALBUMINURIC RETINITIS.

Nettleship, R. L. O. H. Rep., xv. Masses of fibrinous exudate in the retina.

rhages are not at all infrequent here. Special stress is laid upon degeneration of the chorio-capillaries by v. Michel. He regards the retinitis as secondary to the retinal vascular degeneration. v. Graefe, Leber and others regarded the retinitis as secondary to the kidney mischief; v. Michel, Herzog Karl Theodor, Weeks, Greeff and others, regard both as due to a common cause.

In those cases in which the patient lives sufficiently long retinal degeneration reaches a high grade, the normal tissues becoming shrunken and replaced by new connective tissue in the manner common to other conditions.

It has been mentioned that slight detachment of the retina occurs around the disc from subretinal exudates. Leber and Nordenson attribute complete detachment to shrinkage of the vitreous. Kunz considers it due to active accumulation of subretinal fluid as well as vitreous shrinkage. Goldzieher considers that it is caused by the retinal œdema alone, the fluid transuding into the subretinal space.

Of these explanations the last is least probable, but the others are unsatisfactory. It will probably be found eventually that this and some other forms of retinal detachment are due to osmotic conditions of which we at present know nothing.

SCHWEIGGER.—A. f. O., vi, 2, 1860.   LIEBREICH.—Atlas, Pl. ix, 1863.   TREITEL.—A. f. O., xxii, 2, 1876.   PONCET.—Atlas de l'Anat. path. de l'Œil, Paris, 1879.   OELLER.—Virchow's Archiv, lxxxvi, 1881.   NORDENSON.—Die Netzhautablösung, Wiesbaden, 1887.   HERZOG KARL THEODOR.—Ein Beitrag zur path. Anat. des Auges bei Nierenleiden, Wiesbaden, 1887.   WEEKS.—A. f. A., xxi, 1889.   W. J. COLLINS.—T. O. S., ix, 1889.   v. MICHEL.—Lehrbuch, 1890; Z. f. A., ii, 1889.   HAAB.—B. z. A., v, 1892.   DIMMER.—Internat. Med. Congress, Edinburgh, 1894; B. z. A., xiii, 1894.   NUEL.—A. d'O., xv, 1895.   KUNZ.—Dissertation, Marburg, 1897.   NETTLESHIP.—T. O. S., xix, 1899; R. L. O. H. Rep., xv, 4, 1903.   GOLDZIEHER.—Die ophth. Klinik, 1900.   RÖMER.—A. f. O., lii, 3, 1901.   KOPPEN.—Z. f. A., viii, 1902.   HOFMANN.—A. f. A., xliv, 1902.   UHTHOFF.—B. d. o. G., 1903.   GOURFEIN-WELT.—Internat. Ophth. Congress, Lucerne, 1904.   OPIN AND ROCHON-DUVIGNEAUD.—A. d'O., xxiv, 1904.   YAMAGUCHI.—Z. f. A., xi, 1904.   (See also p. 1298.)

Uræmic amaurosis.—Wells (1812) first drew attention to transient amaurosis occurring with dropsy after scarlet fever. Uræmic amaurosis is rarer than albuminuric retinitis, but it is probably often masked by the mental condition of the patient. It occurs paticularly in acute nephritis, e.g. in pregnancy (Greve, Weber, Decoin, Walliter, Szili, Marcuse), after scarlet fever (Foerster, Ebert, Monod, Flögel, Barlow, Becher), smallpox (Adler), etc., but is also seen with chronic nephritis. The onset of blindness is sudden or rapid (8—24 hours) ; it is bilateral and complete. The fundi show no changes (Foerster, v. Graefe, and others). Vision usually improves in from 10—18 hours and is fully restored in about 48 hours, so that the whole process occupies 3—5 days. One eye may improve more rapidly than the other. Uræmic amaurosis may be the first sign of latent nephritis. During the attack the urine is diminished, very rarely increased ; it may be free from albumin during the blindness (Ebert). The pupils are dilated, but usually react to light (Schmidt-Rimpler).

Ophthalmoscopic changes have been present in some cases, but do not explain the complete blindness. Albuminuric retinitis has been seen (v. Graefe, Schmidt-Rimpler), also papillitis (Dobrowolsky, v. Michel, Förster, Kampherstein). Amblyopia as opposed to amaurosis, hemianopia (Köppen, Putzel, Pick, Lehmann), and night-blindness (Knies) have been described. Obstruction of the central artery of the retina has been recorded in three cases, all women (Schmidt-Rimpler).

The disease occurs not only in interstitial but also in parenchymatous nephritis, not in amyloid degeneration, in which uræmia is rare. In the cases during pregnancy there is generally eclampsia (Litzmann, Eliasberg, Marcuse) ; evidence of renal disease may be absent (Decoin). Cases of transient puerperal amblyopia or amaurosis in which no statement as to the condition of the urine is given probably belong to this disease (Ringlard). Some cases of amaurosis during lead poisoning (Hirschler) also belong to this category (Schmidt-Rimpler).

As regards frequency, v. Graefe in 32 cases of amblyopia with albuminuria found ophthalmoscopic changes in 30, none in 2 ; Wagner found 1 case of uræmic amaurosis in 157 cases of nephritis. In 13

cases of eclampsia with nephritis gravidarum Litzmann found the disease 3 times.

The pathogenesis is obscure. Transient œdema of the retina (Heyl), optic nerve (Ebert) or its sheaths (Rothmann) is not supported by the symptoms. Persistence of the pupillary light reaction is strong evidence in favour of a toxic lesion above the " primary " optic centres. Löb saw a case in a child after scarlet fever with persistent paralysis of the left arm. The cause is to be sought in that of the uræmia. Of this there are two chief theories. Traube advanced the opinion that it was due to the high blood-pressure which caused intra-cranial œdema and cerebral anæmia (Vaquez). This mechanical theory involves increased intra-cranial pressure, which the usual absence of papillitis negatives. The chemical theory of v. Frerichs has more in its favour, but the toxic agent remains to be discovered (Bouchard). Whether it acts as a cerebral irritant (Landois) or by producing cerebral anæmia (Fleischer) cannot be discussed here.

Wells.—Tr. Soc. for the Improvement of Med. and Chir. Knowledge, 1812. Greve.— Deutsche Klinik, 1873. Weber.—Berliner klin. Woch., 1873; 1878. Decoin.—Gaz. des Hôp., 1876. Walliter.—St. Louis Med. and Surg. Jl., 1879. Szili.—C. f. A., vi, 1882. Marcuse.—Z. f. klin. Med., xiii, 1888. Foerster.—Jahrb. f. Kinderheilk., v, 1872; in G.-S., vii, 1876. Ebert.—Berliner klin. Woch., 1868. Monod.—Gaz. des Hôp., 1870. Flögel.— Prager med. Woch., 1878. Barlow.—Med. Times and Gaz., 1881. Becher.—Deutsche med. Woch., 1884. Adler.—Die wahrend u. nach. d. Variola auftretenden Augenkrank-heiten, Wien, 1874. v. Graefe.—A. f. O., ii, 2, 1858. Schmidt-Rimpler.—Berliner klin. Woch., 1870; Die Erkrankungen, etc., Wien, 1905. Dobrowolsky.—K. M. f. A., xix, 1881. v. Michel.—A. f. O., xxiii, 2, 1877. Kampherstein.—K. M. f. A., xliii, 1905. Köppen.— Charité Annalen, 18 Jahrgang. Putzel.—Med. Rev., 1888. Pick.—Deutsches A. f. klin. Med., 1895. Lehmann.—Berliner klin. Woch., 1896. Knies.—Die Beziehungen, etc., Wiesbaden, 1893. Eliasberg.—C. f. A., xvii, 1893. Litzmann.—Deutsche Klinik, 1852. Ringlard.—In Stellwag v. Carion, Die Ophthalmologie vom naturw. Standpunkt, 1855. Hirschler.—Wiener med. Woch., 1866. Wagner.—Virchow's Archiv, xii, 1857. Heyl.— Am. Jl. of Med. Sc., 1874. Rothmann.—Berliner klin. Woch., 1894. Löb.—Jahrb. f. Kinderheilk., viii, 1875. Vaquez.—Tribune méd., 1904. Hargreaves.—Brit. Med. Jl., 1884. Wilkinson.—Am. Jl. of O., 1897. Bouchard.—Les Auto-intoxications, Paris, 1887. Pal.—Centralbl. f. innere Med., 1903. Hauenschild, Roscher, Pick.—Münch. med. Woch., 1903.

Other ocular changes in renal disease may be briefly mentioned. Œdema of the lids is common. Spontaneous conjunctival hæmorrhages may occur with albuminuric retinitis and may precede it (Talko, Hirschberg). Wharton Jones reports hæmorrhage into Tenon's capsule. Primary inflammation in the uveal tract has been reported—iritis (Leber, Knies, Schapringer, Nettleship), choroiditis (Liebreich, Magnus, Schreiber). Choroidal changes are common in sections of albuminuric retinitis, including vascular changes, hæmorrhages (Weeks, Nettleship, and others), but the ophthalmos-copic changes ascribed to the choroid are probably chiefly retinal. Interstitial keratitis has been reported in two cases by Eversbusch, and keratitis, glaucoma, and panophthalmitis by Pollack. Albuminuria as a factor in the pathogenesis of cataract has already been mentioned (v. Vol. III, p. 1024). Renal disease and its results as factors in the production of many other ocular conditions, e.g. glaucoma, retrobulbar neuritis, paralysis of extrinsic muscles, etc., have been referred to incidentally.

HIRSCHBERG.—Deutsche Z. f. prakt. Med., 1876. WHARTON JONES.—Brit. Med. Jl., 1863. LEBER.—A. f. O., xxxi, 4, 1885. SCHAPRINGER.—Am. Jl. of O., 1893. NETTLE-SHIP.—R. L. O. H. Rep., xv, 4, 1903. LIEBREICH.—A. f. O., vi, 2, 1860; Atlas, Pl. xi. MAGNUS.—Die Albuminurie in ihre ophthalmoscop. Erscheinungen, 1873. SCHREIBER.—Deutsches A. f. klin. Med., 1878. WEEKS.—A. of O., xvii, 1888. POLLACK.—Am. Jl. of O., 1893. *GROENOUW.—In G.-S., xi, 1, 1900 (Bibliography).

DISEASES OF THE GENERATIVE ORGANS.

Gonorrhœa.—The ocular complications of gonorrhœa, which are referred to in greater detail elsewhere (*v.* Vol. I, p. 41), may be briefly enumerated here. The most important are the gonorrhœal conjunctivitis of adults, ophthalmia neonatorum, metastatic gonorrhœal conjunctivitis, and gonorrhœal iritis. Rarer complications which have been described are ring infiltration of the cornea with hypopyon (Martin), retinitis (Hilbert), neuroretinitis (Campbell), and dacryoadenitis (Pes, Terson).

*STEPHENSON. — Ophthalmia Neonatorum, London, 1907. *Gonorrhœal iritis.*—LAWRENCE.—Venereal Diseases of the Eye, London, 1830. HUTCHINSON.—R. L. O. H. Rep., vii, 1873. RÜCKERT.—K. M. f. A., xxiv, 1886. NOBEL.—A. f. Derm. u. Syphilis, xxiii, 1894. LICHTENSTEIN.—Prager med. Woch., 1898. *Rare complications.*—COLSMANN.—Berliner klin. Woch., 1882. MARTIN.—A. f. A., xiv, 1882. CAMPBELL.—Ann. d'Oc., cxv, 1896. HILBERT.—Z. f. prakt. Aerzte, 1896. PES.—Die ophth. Klinik, 1898. TERSON.—Die ophth. Klinik, 1900. HOCHEISEN.—A. f. Gynäk., lxxix, 1906. ROOSA.—Postgraduate, 1906.

Soft chancre.—In sixty-six cases of soft chancre on the head and face Eudlitz found three on the lids. It resembles vaccinia of the lids. Koeber succeeded in inoculating rabbits' eyelids with virus from a human soft chancre.

KOEBER.—Wiener med. Woch., 1883. VIGNES.—Ann. d'Oc., cx, 1894; cxx, 1899. EUDLITZ.—Arch. gén. de Méd., 1897. ROTHENPIELER.—C. f. A., xxii, 1898.

Masturbation and sexual intercourse.—Masturbation has been held responsible for photopsiæ (Cohn), photophobia (Mooren), amblyopia (Dieu, Glascott, Hutchinson), conjunctivitis (Foerster, Landesberg, Cohn), cycloplegia (Mooren, Landesberg), etc. Similar results have been attributed to sexual intercourse, and this cause may be undoubtedly responsible for retinal hæmorrhage (Knies) in the predisposed, proptosis (Foerster), etc.

DIEU.—Jl. d'O., i, 1872. HUTCHINSON.—R. L. O. H. Rep., ix, 1876; Arch. of Surg., iv, 1900. LANDESBERG.—Med. Bull., 1881. COHN.—A. f. A., xi, 1882. GLASCOTT.—Ophth. Rev., ii, 1883. POWER.—T. O. S., vii, 1887. SPALDING.—T. Am. O. S., 1897. MOOREN.—Gesichtsstörungen u. Uterinleiden, Wiesbaden, 1898.

Various ocular disorders have been attributed to disease of the female organs of generation since the time of Hippocrates. The subject has been much discussed, but it is one of those borderland questions which is apt to be insufficiently investigated. Many of the observations are comparatively worthless, and further research would probably elicit valuable information.

v. GRAEFE.—A. f. O., xii, 2, 1866. SWANZY.—Obstet. Jl., ii, 1878. GEORGEON.—Thèse, Paris, 1880. MOOREN.—A. f. A., x, 1881; Gesichtsstörungen u. Uterinleiden, Wiesbaden, 1898. FITZGERALD.—Lancet, 1883. POWER.—T. O. S., viii, 1888. KOLLOCK.—Trans. South

Carolina Assoc., Charlestown, 1888. S. Cohn.—Uterus u. Auge, Wiesbaden, 1890. Bataud. —Rev. des Maladies des Femmes, xii, 1890. Berger.—Les Maladies des Yeux dans leurs Rapports avec la Path. gén., Paris, 1892 ; Encyclop. franç. d'O., iv, 1905. *Berger and Loewy.—Ueber Augenerkrankungen sexuellen Ursprungs bei Frauen, Wiesbaden, 1906 (Bibliography). Bettmann.—Am. Obstet. Jl., xxviii, 1893. Ladlam.—New York Med. Times, 1893. Ramsay.—Lancet, 1893. Wood and Woodruff.—New Am. Practitioner, 1894. Derby.—New York Eye and Ear Infirmary Rep., 1898. Terrien.—Gaz. des Hôpi- taux, 1903.

Puberty.—Puech states that ocular diseases reach their maximum at puberty, but Schleich found the two periods of greatest frequency between sixteen and twenty and between fifty-six and sixty.   The onset of menstruation may have a beneficial effect upon interstitial keratitis (Mooren, Dunn) ; on the other hand, like injury, it may be the exciting cause of an attack (Cohn).   Uveitis is said to be commoner at puberty (Pflüger, Dauthon, Pressel, Hiram Woods), and several cases of vitreous hæmorrhage are recorded (Pressel, Dor, Coursserant).   Cases of optic neuritis and atrophy, sometimes associated with deafness, with uterus infantilis and delayed menstruation are reported by Leber, Beer, Rockcliffe, Berger and Loewy; hæmorrhage into the nerve sheath (Schmidt-Rimpler), and toxæmia (Berger and Loewy) are cited as the cause.   Improvement of vision (Brièrre de Boismont, Santos Fernandez), and diminution of vision (Bock), both probably due to hysteria, may accompany the onset of the menses.

Puech.—Rec. d'O., 1889. Schleich.—Bericht d. Augenklinik in Tübingen (1875— 1901). Dunn.—A. of O., xix, 1895. Hiram Woods.—Jl. of Eye, Ear, and Throat Diseases, 1896. Beer.—Wiener klin. Woch., 1892. Rockliffe.—T. O. S., xxiv, 1904. Santos Fernandez.—Jahrb. d. Augenheilkunde, 1879. Bock.—Allg. Wiener med. Zeitung, 1890.

Normal menstruation. — Normal menstruation may certainly aggravate ocular disease already present, and there are grounds for thinking that it may give rise to ocular complications in debilitated women.   Amongst the disorders directly ascribed to menstruation are coloration of the lids, œdema of the lids (Boerner), hordeolum (Dianoux, Galezowski, Pflüger), hæmorrhage into the lids (Dolganow), conjunctivitis (Friedenwald, Berger and Loewy, Müller, Seeligsohn) conjunctival hæmorrhage (Perlia), herpes corneæ (Ransohoff, Landesberg, Stuelp), hæmorrhage into the anterior chamber (Jüngken, Landesberg), vitreous hæmorrhage (Friedenwald), contraction of the field of vision (Finkelstein), retinal hæmorrhage and papillitis (Leber, Hinzinga), amaurosis (Christensen, Meyer), muscular asthenopia, etc. An attack of glaucoma may be indirectly induced by normal menstrua- tion.   Hysteria and other nervous disorders may affect the vision at this period.   Normal menstruation may aggravate diseases already present, e. g. elephantiasis of the lids (Fage), chronic conjunctivitis (Dianoux, Wengler, Berger and Loewy), herpes corneæ, interstitial keratitis (Mooren, Berger and Loewy), phlyctenular keratitis, episcleritis (Mooren), iritis (v. Michel, Klopstock, Despagnet, de Wecker, Bataud and Vignes), iridochoroiditis (Pflüger, Trousseau), optic neuritis (Leber), quinine amblyopia (Grüning), exophthalmos (McKay).   The effect of menstruation is ascribed to circulatory changes (Groenouw), and to toxæmia (Trousseau, Janot, Pargoire, Berger and Loewy).

BOERNER.—Volkmann's Sammlung, cxxxii.   DOLGANOW.—St. Petersb. med. Woch.,
1900.   FRIEDENWALD.—Jl. of Eye, Ear, and Throat Diseases, 1896.   MÜLLER.—K. M. f. A.,
xxxii, 1893.   SEELIGSOHN.—C. f. A., xx, 1896.   PERLIA.—Münchener med. Woch., 1888,
RANSOHOFF.—K. M. f. A., xxvii, 1889.   LANDESBERG.—C. f. A., vii, 1883.   STUELP.—
A. f. O., xl, 2, 1894.   FINKELSTEIN.—Wratsch, 1886.   VANCE.—Boston Med. and Surg. Jl.,
1872.   LEBER.—In G.-S., v, 1877.   HINZINGA.—Jl. Am. Med. Assoc., 1902.   MEYER.—
Berliner klin. Woch., 1874.   HIRSCHBERG.—Berliner klin. Woch., 1872.   H. COHN.—
K. M. f. A., v, 1867.   FAGE.—Ann. d'Oc., cvii, 1892.   WENGLER.—Jl. f. Chir., viii, 1898.
DECKER.—K. M. f. A., xxxviii, 1900.   TROUSSEAU.—A. de Toxicologie, xvii, 1890.
DESPAGNET.—Soc. franç. d'O., 1891.   BATAUD AND VIGNES.—A. d'O., xvi, 1896.   GRÜNING.—
A. f. A., xi, 1882.   McKAY.—Am. Jl. of Med. Sc., 1882.

Dysmenorrhœa.—Dysmenorrhœa may be due to disease of the sexual
organs or to general disease.   In the former case it may be associated
with episcleritis and scleritis (Saemisch, Mooren), hyphæma (Coursserant,
Jüngken), iritis (Abadie), iridocyclitis (Guépin, Mooren), iridochoroditis
(Mooren, Caudron, Terrien), vitreous hæmorrhage (Berger and Loewy),
papillitis (v. Graefe, Swanzy, Foerster), retrobulbar neuritis (Uhthoff),
paralysis of extrinsic muscles, subjective defects of vision, etc.   Dys-
menorrhœa may be due to other diseases and cause ocular manifesta-
tions, *e. g.* tuberculosis, anæmia, hysteria, etc.   Chromhidrosis may
occur under these conditions (Rothmund), and xanthopsia is recorded
(Kohn).

SAEMISCH.—In G.-S., iv, 1876.   GUÉPIN.—Ann. d'Oc., xlvi, 1861.   SWANZY.—Irish
Hosp. Gaz., 1873.   UHTHOFF.—A. f. O., xxxiii, 1, 1887.   v. HASNER.—Prager med. Woch.,
1883.   WINGENROTH.—A. f. A., liv, 1906.   LÖWENFELD.—Sexualleben u. Nervenleiden,
Wiesbaden, 1903.   ROTHMUND.—K. M. f. A., v, 1867.   KOHN.—Rec. d'O., 1873.

Amenorrhœa.—Amenorrhœa, like dysmenorrhœa, may be due to
disease of the sexual organs or to general disease.   Of the latter
acromegaly and cerebral tumours are specially to be borne in mind.
Amenorrhœa may be associated with " bloody tears" (Jüngken,
Hasner, Heusinger), phlyctenules (Coursserant), interstitial keratitis
(Mooren), hyphæma (Lawrence), vitreous hæmorrhage (Friedenwald,
Davis, Hotz, Bylsma), disseminated choroiditis (Mooren), retinal
hæmorrhages (Liebreich, Friedenwald), optic neuritis (Mooren, Meyer,
Ewers), optic atrophy (Galezowski, Christensen, Berger and Loewy)—
special care to eliminate cerebral tumour is necessary (*cf.* Karafiath,
Schmidt-Rimpler, Axenfeld, Yamaguchi, Bayerthal, Abelsdorff, L.
Müller, Herbst)—retrobulbar neuritis (Rampoldi, Abelsdorff).

GALEZOWSKI.—Rec. d'O., 1875.   ABELSDORFF.—A. f. A., xxi, 1890.   v. HASNER.—
Wiener med. Woch., 1859.   DAVIS.—Ophth. Rec., 1897.   HOTZ.—Annals of O., 1893.
BYLSMA.—Münchener med. Woch., 1902.   LIEBREICH.—Atlas, Pl. viii, fig. 2, 1863.
FRIEDENWALD.—T. Am. O. S., 1903.   RAMPOLDI.—Ann. di Ott., xiv, 1885.   AXENFELD.—
Vereinigung Südwestdeutscher Irrenärzte, 1902.   YAMAGUCHI.—K. M. f. A., xli, Beilageheft,
1903.   HERBST.—Wiener klin. Woch., 1902.   WYGODSKY.—In Nagel's Jahresbericht, 1904.

Suppressio mensium.—Sudden suppression of menses may be accom-
panied by corneal infiltration or ulceration (Daguenet, Teillais), iritis
(Thaon, Teillais), hyphæma (Cohn), vitreous hæmorrhage, disseminated
choroiditis (Machek), optic neuritis or atrophy, amaurosis (Brown,
Skorkowski and Kofminski, Samelsohn, Mooren, Stocker, Galezowski,
McKay, Sutphen, Chiralt, Ruete), retinal hæmorrhage (Coursserant),
paralysis of extrinsic muscles (Mooren, McKay), etc.   The pathogenesis

of these disorders is doubtful. Foerster and Leber attribute some to hyperæmia of the optic nerve, Mooren to derangement of the occipital lobes by venous obstruction in the sinuses, Terrien to infection ; the toxæmia theory (Berger and Loewy) is perhaps the most probable.

Daguenet.—Rec. d'O., 1876. Machek.—Rev. gén. d'O., 1881. Samelsohn.—Berliner klin. Woch., 1874. Sutphen.—T. Am. O. S., 1891. Chiralt.—Ann. d'Oc., lxxiii, 1875. Berger and Loewy.—Ueber Augenerkrankungen sexuellen Ursprunges bei Frauen, Wiesbaden, 1906.

Climacteric.—Ocular complications occurring during the climacteric are conjunctivitis and conjunctival hæmorrhage (Evans), episcleritis (Berger and Loewy), iritis (Sichel), cyclitis (Middlemore), glaucoma (Naumann and others), intra-ocular hæmorrhage (Terrien), primary optic atrophy (Berger and Loewy), optic neuritis (Stocke, Freund, Leber). The climacteric often aggravates ocular disturbances due to nervous diseases, *e.g.* hysteria.

Evans.—Am. Med., 1903. Naumann.—Balneologische Zentralzeitung, 1902. Stocke.—La Clin. ophth., 1902.

Insufficiency of the ovaries.—Chlorosis (q. v.) and Dercum's disease have been attributed to abnormality or diminution of the ovarian internal secretion. In the latter disease Berger and Loewy have found subjective disturbances of vision. Amblyopia or amaurosis have been recorded after castration (Jayle, Collins, Berger).

Jayle.—Rev. de Gynécologie, 1897. Collins.—Lancet, 1886. Berger.—A. d'O., xxvii, 1897. Caudron and Duboys de Lavigerie.—Ann. d'Oc., cxxix, 1903. Culbertson.—Am. Jl. of O., xiv, 1897.

Diseases of the female generative organs.—Apart from the disorders already considered diseases of the uterus, etc., may give rise to ocular complications. Most of those recorded are subjective disturbances of vision. The occurrence of episcleritis in the course of uterine disease is too frequent to be fortuitous (Saemisch, Mooren). Iritis also occurs with endometritis (de Wecker, Bataud and Vignes) : I have seen severe scleritis in one eye followed by iridocyclitis in the other. Mydriasis, (Mannhardt), cycloplegia (Berger and Loewy) are described, and trigeminal neuralgia is not uncommon (Groenouw). Glaucoma may be precipitated by retroflexion of the uterus (Wicherkiewicz) or puncture of an ovarian cyst (Foerster). Metastasis from carcinoma uteri may be choroidal (v. Vol. II, p. 533) or in the extrinsic muscles (Elschnig). Metastases in both intervaginal spaces from ovarian carcinoma have been reported (Krohn). Reflex hyperæsthesia of the trigeminal and optic nerves in uterine disorders has been described as kopiopia hysterica (Foerster).

Wilbrand and Saenger.—Ueber Sehstörungen bei func. Nervenleiden, Leipzig, 1892 ; Neurologie des Auges, Wiesbaden, 1900—   . Mannhardt.—K. M. f. A., xxv, 1887. Elschnig.—Wiener klin. Woch., 1898. Krohn.—K. M. f. A., x, 1871. Foerster.—In G.-S., vii, 1877.

Pregnancy.—According to Winckel 1·6 per cent. of pregnant women have ocular disturbances. Many are reflex in origin. The relative importance·of changes in the circulation and blood, auto-intoxication

(Charrin and Roché, Planchu) due to defective metabolism (Charrin and Jardry), gastro-intestinal fermentation, fœtal or placental toxins, defective anti-toxic action of the liver (Rogers), changes in internal secretion of ductless glands (Charrin and Guiyesse), suppression of menses, insufficiency of ovarian secretion (Charrin and Guillemonat), etc., must be left an open question. Pregnancy may be accompanied by pigmentation of the lids, hyper-secretion of tears (Nieden), corneal ulcers, conical cornea (Valude), conjunctival hæmorrhage (Guttmann), mydriasis (Fisher), cycloplegia, choroiditis and retinal detachment (Delzoppo and Soli), glaucoma (Galezowski, Landesberg), cataract (Power, Pflüger, Terrien), contraction of field and subjective disturbance of vision (Bellinzona and Tridondani, Schoen, Knaggs), night-blindness (Küstner and others), amaurosis with icterus (Lutz, Landesberg), optic neuritis (Tissier, Knapp, Bull, Bar, Reich), retrobulbar neuritis (Mackenzie, Uhthoff, Level), retinal hæmorrhage (Teillais, Cohn), pulsating exophthalmos (Sattler), etc. More important are the cases with renal disease (v. p. 1296).

Winckel.—Berichte a. d. Kgl. Sächsischen Entbindungs-Institut in Dresden, 1876. Charrin and Roché.—Comptes rendus, 1903. Planchu.—Gaz. des Hôpitaux, 1904. Nieden.—K. M. f. A., 1901. Guttmann.—C. f. A., xxviii, 1904. Fisher.—Ophth. Rev., xvi, 1898. Delzoppo and Soli.—Ann. di Ott., xxxiii, 1904. Landesberg.—A. f. O., xxiv, 1, 1878. Power.—Lancet, 1880. Hoppe.—A. f. O., lvi, 1903. Küstner.—Berliner klin. Woch., 1875. Lutz.—Mitt. a. d. ophth. Klinik in Tübingen, 1884. Bosse.—A. f. A., xlii, 1900. Reich.—K. M. f. A., xx, 1882. Uhthoff.—A. f. O., xxxiii, 1, 1887 ; Berliner klin. Woch., 1890 ; Internat. Med. Congress, Paris, 1900. Level.—Ann. d'Oc., xix. Tissier.—Soc. d'Obstet. de Paris, 1904. Knapp.—Brit. Med. Jl., 1893. Knaggs.—T. O. S., xvi, 1896. Bull.—Ann. d'Oc., cviii, 1892. Teillais.—Ann. d'Oc., xcv, 1886. Sattler.—In G.-S., vi, 1880. Lawson.—R. L. O. H. Rep., iv, 1863. Galezowski.—Rec. d'O., 1874. Metaxas.—Rec. d'O., 1883. Rampoldi.—Ann. di Ott., xiv, 1885. Valude.—Ann. d'Oc., cvii, 1892. Delzoppo and Soli.—Ann. di Ott., xxxiii, 1904. Polte.—K. M. f. A., xliii, 1905. Kipp.—Ophth. Rec., 1906.

Parturition.—The pupils are dilated during parturition (Raehlmann and Witkowsky). Hæmorrhage into the conjunctiva, vitreous (Schmidt-Rimpler), orbit (Boehm), may occur, and great loss of blood may lead to characteristic ocular symptoms (v. p. 1316). Transitory amblyopia or amaurosis occurs even in the absence of eclampsia (Weber, Königstein, Matheson, Hirschler) ; Hirschler found no ophthalmoscopic changes.

Weber.—Berliner klin. Woch., 1873. Hirschler.—Wiener med. Woch., 1874. Raehlmann and Witkowsky.—A. f. Anat. u. Physiologie, 1878. Königstein.—Wiener med. Presse, 1885. Matheson.—Med. and Surg. Rep., 1886. Boehm.—Die ophth. Klinik, 1899.

Puerperium.—Apart from the ocular complications due to puerperal fever (v. Chap. XXIV) the following may occur : Keratomalacia (Schmidt-Rimpler), paresis of accommodation (Berger and Loewy), choroiditis (Pflüger), uræmic amaurosis (q. v.) (Weber, Mandelstamm), albuminuric retinitis (q. v.) (v. Graefe, Schmidt-Rimpler, Panas), hysterical amblyopia (Weber, Szili, Eastlake), xanthopsia (Davis), retrobulbar neuritis (Pflüger, Saenger, Reuling), retinal hæmorrhage (Wernicke and Küstner), embolism of the central artery (Nagel, Walter), occipital embolism (Pflüger), etc.

v. Graefe.—A. f. O., ii, 1, 1855. Wernicke and Küstner, Weber.—Berliner klin. Woch., 1873. Reuling.—New York Med. Jl., 1877. Mandelstamm.—St. Petersb. med. Woch., 1878. Pflüger.—A. f. O., xxiv, 2, 1878 ; in Cohn, *loc. cit.* Walter.—Brit. Med. Jl., 1881. Nagel.—C. f. A., v, 1881. Szili.—C. f. A., vi, 1882. Badal.—A. d'O., viii, 1888. Cohn.—Uterus u. Auge, Wiesbaden, 1890. Lehmann.—Berliner klin. Woch., 1896. Saenger.—Mitt. a. d. Hamburger Krankenanstalt, 1897. Silex.—Monatsschrift f. Geburt- shilfe u. Gynäkologie, v, 1897. Zimmermann.—A. of O., xxvii, 1898.

Lactation.—Ocular concomitants of lactation which have been recorded are conjunctivitis (Middlemore, Schröder), phlyctenular ophthalmia (Nasse, v. Arlt), herpes corneæ (Godo), mydriasis (Rogman), paresis of accommodation (Hutchinson, Collins, Jacobson, Berger and Loewy), choroiditis and complicated cataract (Mooren), night blindness (Schröder, Leber), retinitis (Mackenzie), optic neuritis (Axenfeld, Heinzel, Rogman, Derby, Lobel, Schanz, Gibbon), amblyopia (Nettle- ship, Lobel), paresis of extrinsic muscles, etc. Retrobulbar neuritis may occur only after weaning (Schmidt-Rimpler). Many of these disorders are probably merely intercurrent, and in any case the exact pathogenesis is purely conjectural.

Middlemore.—Treatise, London, 1835. Carron du Villards.—Traité, Bruxelles, 1838. Nasse.—v. Ammon's Monatsschrift, iii, 1840. Mackenzie.—Treatise, London, 1835. Critchett.—Med. Times and Gaz., 1858. Hutchinson.—R. L. O. H. Rep., vii, 1871. Pflüger.—A. f. O., xxiv, 2, 1878. Godo.—Rec. d'O., 1880. Nettleship.—T. O. S., iv, 1884 ; R. L. O. H. Rep., xiii, 1891. Collins.—Lancet, 1886. Heinzel.—B. z. A., ii, 1894 ; iii, 1895. Rogman.—Ann. d'Oc., cxii, 1894. Axenfeld.—Monatsschrift f. Geburtsh u. Gyn., 1895. Schanz.—Deutsche med. Woch., 1896. Bistis.—A. d'O., xxiv, 1904. Derby.—A. of O., xxxiv, 1905. Lobel.—Rec. d'O., xxviii, 1906.

Constitutional Diseases.

Diabetes mellitus.—Defective vision was first attributed to diabetes by Renauldin (1814). Diabetic cataract was described by Berndt (1834). Diabetic amblyopia, subsequently confirmed by Liman (1842), Ruete (1843), Bouchardat (1846) and Desmarres (1858), was long considered doubtful, but Jaeger (1856) published the first ophthalmo- scopic demonstration of diabetic retinitis and the matter was finally set at rest by Leber (1875). Paralysis of accommodation in diabetes was first described by v. Graefe (1858), who considered that many of the cases of amblyopia were due to this cause.

As regards the frequency of ocular complications in diabetes Seegen found them in two thirds of the cases, which is certainly too high (Leber). Estimates are given by Schmidt-Rimpler, Galezowski, Lagrange, König, Hirschberg, Williamson, and others. The most important are as follows : Cataract, in all diabetics, 2 per cent. (König), 5 per cent. (v. Frerichs), 8 per cent. (Williamson) ; in diabetics with eye disease, 18 per cent. (König), 25 per cent. (Lagrange), 45 per cent. (Schmidt-Rimpler) ; retinitis, in all diabetics, 6·5 per cent. (Williamson) ; in diabetics with eye disease, 23 per cent. (Schmidt-Rimpler), 36·5 per cent. (Lagrange). The following statistics are in diabetics with eye disease : affection of optic nerve, 6 per cent. (Lagrange), 25 per cent. (Schmidt-Rimpler) ; iritis or choroiditis, 5 per cent. (Schmidt-Rimpler), 6 per cent. (Lagrange) ; paralysis of extrinsic muscles, 2 per cent. (Lagrange), 7 per cent. (Schmidt-Rimpler). The statistics of

amblyopia without ophthalmoscopic signs, cycloplegia, vitreous opacities etc., are little trustworthy; keratitis (Galezowski, Lagrange), and palpebral or orbital abscess (Lagrange) are reported.

Ocular complications may occur at any age but is commonest in advanced age (youngest 9 years, 27 per cent. between 50 and 60, 35 per cent. between 60 and 70, Galezowski). They are commoner in men than in women (65 per cent. Galezowski, 71 per cent. Seegen, 79 per cent. Schmidt-Rimpler).

The prognosis as to life is difficult to estimate; the not infrequent association of albuminuria complicates the question, and still more so the ætiological varieties of diabetes mellitus, transient slight glycosuria and the slight glycosuria of old age being frequently of little importance *quoad vitam*. It is certain that true diabetes can persist long without ocular symptoms, and also that the prognosis is not so bad in diabetic as in albuminuric retinitis (q. v.). Papanikolau found that of 41 diabetics with eye disease 19 died within 2 years; a case of retinitis lived $3\frac{1}{4}$ years, one of central choroiditis 4 years, one of neuritis 5 years, 2 with cataract—a man æt. 66, and a woman æt. 63— 7 years, and a girl æt. 10 with cataract 19 years. Hirschberg and Schirmer regard optic nerve affection as of malign import, Schmidt-Rimpler as not unfavourable.

Diabetic cataract.—See Vol. II, p. 425; Vol. III, p. 1019.

Diabetic retinitis resembles albuminuric retinitis in the presence of hæmorrhages and white spots. Nettleship found it much commoner in private than in hospital patients (fifty-two out of fifty-nine cases). In many the urine contains some albumin, but there is no doubt that retinitis may be due to diabetes without albuminuria (Stephen, Mackenzie, Anderson, Hirschberg, Juler, Rockcliffe, Nettleship). Dimmer found maltose in the urine in one case, not glucose. Diabetic retinitis differs ophthalmoscopically from the renal form in the absence of the soft-edged or wooly patches and of the stellate arrangement of the white deposits around the yellow spot; moreover there is usually no œdema. The hæmorrhages are more often round and deeply seated than linear and superficial as in renal retinitis (Nettleship); in diabetes they are often derived from capillary aneurysms, not from rupture of larger vessels in the nerve-fibre layer. The white deposits take the form of irregular masses or clumps rather than dots or round patches, and are often arranged in a rude zone or ring at the circumference of the macular area. In a few instances hæmorrhages into the retina and vitreous form the chief feature. The disease is nearly always bilateral, but the second eye may be affected late (Reik), and rarely hæmorrhages are confined to one eye for years (Schmidt-Rimpler). Thrombosis of the central vein (q. v.) (v. Michel, Hirschberg), embolism of the central artery (q. v.) (Knapp, Dodd), hæmorrhagic glaucoma (Galezowski, Knapp, Hirschberg, König, Leber), etc., occur. Hæmorrhages not infrequently lead to the formation of new vessels in and upon the retina (Nettleship and others) or to extensive retinitis proliferans (q. v.) in this disease. Detachment of the retina occasionally occurs.

The ophthalmoscopic signs of disease of the vessel walls, so promi-

nent a feature of renal retinitis, are usually absent, and papillitis is rare.

A peculiar feature sometimes met with in diabetes is lipæmia. The ophthalmoscopic appearances are then striking, the retinal vessels containing fluid which looks like milk (Heyl, Hale White, Fraser, Heine). The arteries are pale reddish, the veins having a slight violet tint. The general fundus has much the normal coloration; Uhthoff suggests that this is due to the lighter fat globules arranging themselves along the walls of the vessels, the heavier red corpusles remaining in mid-stream. Heine found diminution of the intra-ocular tension in diabetic coma.

As regards prognosis to life Nettleship traced the history in 48 cases. Of these patients, aged from 41 to 79 years, 38 died—9 within 1 year, 11 during the second year, 18 between 2 and 8 years; 10 are known to have been living 2 to 10 years after the discovery of the retinitis. Thus 60 per cent. lived more than 2 years, so that the prognosis is much better than in renal retinitis. Most of the diabetics are over 50 years of age when retinitis occurs and the better prognosis is to be ascribed to the nature of the disease. In patients over 55, of 24 with renal retinitis one third survived for 2 years, of 38 with diabetic retinitis more than half lived 2 years or more. The prognosis is rather worse if the disease arises after 55 than before. In 24 fatal cases there was diabetic coma in 11, gangrene in 3, acute uncontrollable diarrhœa in 1, and sudden heart failure in several. These results discount the importance of slight albuminuria, though in 3 cases there was doubt whether the coma was uræmic or diabetic.

The anatomical changes are similar to those found in renal retinitis, but most of the cases examined have been of long duration or of complicated type (e. g. Leber and Hummelsheim). In some hyaline degeneration of the vessels (Nettleship in Mackenzie's case), etc., was a marked feature.

GALEZOWSKI.—Rec. d'O., 1873. HALTENHOFF.—K. M. f. A., xi, 1873. LEBER.—In G.-S., v, 1877; A. f. O., xxxi, 4, 1885. STEPHEN MACKENZIE.—R. L. O. H. Rep., ix, 1877. KNAPP.—A. f. A., x, 1880. *NETTLESHIP.—Med. Times and Gaz., 1885; T. O. S., ii, 1882; vi, 1886; viii, 1888; R. L. O. H. Rep., xv, 4, 1903; xvi, 1, 1904. SAMELSOHN.—Deutsche med. Woch., 1885. HIRSCHBERG.—C. f. A., x, 1886; xv, 1891. ANDERSON.—Ophth. Rev., viii, 1889. SCHWEIGGER.—Deutsche med. Woch., 1891. SEGGEL.—Münchener med. Woch., 1891. JULER.—T. O. S., xii, 1892. DAHRENSTÄDT.—C. f. A., xvi, 1892. ROCKLIFFE.—T. O. S., xiv, 1894. DODD.—A. f. A., xxxi, 1895. ALT.—Am. Jl. of O., 1896. REIK.—Ann. of O., 1899. DIMMER.—K. M. f. A., xxxix, 1901. LEBER AND HUMMELSHEIM.—A. f. O., lii, 1901. ORLANDINI.—Ann. di Ott., xxxi, 1902. HEYL.—T. Am. O. S., 1880. REIS.—A. f. O., lv, 3, 1903. KAKO.—K. M. f. A., xli, 1903. HALE WHITE.—Lancet, 1903. FRASER.—Scottish Med. and Surg. Jl., 1903. *HEINE.—K. M. f. A., xliv, 1906.

Diabetic retrobulbar neuritis belongs to the group of toxic amblyopias (q. v.). Primary optic atrophy (Zirm) is very rare if it occurs at all (Leber, Schmidt-Rimpler, Uhthoff).

FITZGERALD.—Dublin Quart. Jl. of Med. Sc., 1870. GROSSMANN.—Berliner klin. Woch., 1879. NETTLESHIP AND EDMUNDS.—T. O. S., i, 1881; Med. Times and Gaz., 1882; T. O. S., iv, 1884. LAWFORD, MORTON.—Med. Times and Gaz., 1882. SAMELSOHN.—A. f. O., xxviii, 1, 1882. SAMUEL.—C. f. A., vi, 1882. COHN.—A. f. A., vii, 1878. MAUTHNER.—Internat. klin. Rundschau, 1893. FRASER AND BRUCE.—Edin. Med. Jl., 1896. SCHMIDT-RIMPLER.—B. d. o. G., 1896. V. GROSZ.—C. f. A., xxii, 1898. UHTHOFF.—Internat. Med. Congress, Paris, 1900. ZIRM.—C. f. A., xxv, 1901.

Other ocular complications of diabetes.—Homonymous hemianopia has been described by v. Graefe, Galezowski, Wilbrand, Schmidt-Rimpler, and Leber. Paralysis of extrinsic and intrinsic muscles has been frequently reported (see Groenouw). Diabetic hypermetropia (Gallus, Lichtenstein) and myopia have already been discussed (v. Vol. III, p. 929), also the changes in the retinal pigment epithelium of the iris (v. Vol. I, p. 320; Fig. 302).

HORNER.—K. M. f. A., xii, 1873. COHN.—A. f. A., vii, 1878. GRIMSDALE, DOYNE.—T. O. S., xix, 1899. GROENOUW.—In G.-S., xi, 1, 1902; Vossius' Sammlung, vii, 1906. ALEXANDER.—K. M. f. A., xli, 1903. GALLUS.—Z. f. A., xv, 1906. LICHTENSTEIN.—Z. f. A., xvi, 1906.

Diabetes insipidus.—Cataract (van der Heyden, Verneuil, Schmidt-Rimpler), paralysis of extrinsic muscles (Gayet, Wiethe, Dreschfeld), homonymous hemianopia (Schön, Becker), bitemporal hemianopia (v. Graefe, David, Spanbok and Steinhaus), optic neuritis and atrophy (Jones, Raymond, Spicer), etc., have been reported in this disease.

v. GRAEFE.—K. M. f. A., iii, 1865. SCHÖN.—Die Lehre vom Gesichtsfeld, Berlin, 1874. HANDFIELD JONES.—Med. Times and Gaz., 1875. GAYET.—Rec. d'O., 1876. STEVENS.—T. Am. O. S., 1878. DRESCHFELD.—C. f. A., iv, 1880. SPANBOK AND STEINHAUS.—Deutsche med. Woch., 1898. HOLMES SPICER.—T. O. S., xx, 1900. PRIBRAM.—Deutsches A. f. klin. Med., lxxvi, 1905.

Oxaluria, phosphaturia.—Retinal and vitreous hæmorrhages may occur in the former complaint (Mackenzie, Leber); soft cataract in young patients (Dor), hemianopia (Coursserant), and retinal hæmorrhages (Trousseau) in the latter.

MACKENZIE.—Ann. d'O., liii, 1865. LEBER.—A. f. O., xxi, 3, 1875; in G.-S., v, 1877. DOR.—Internat. Med. Congress, Geneva, 1877. TROUSSEAU.—Bull. méd., 1897.

Gout.—It is difficult and often impossible to determine the correct ætiology of ocular conditions occurring in gouty people. That the diathesis is the cause of some of these conditions can scarcely be doubted, but it is still more probable that many are due to intercurrent disease and are modified by the constitutional disorder. In the present unsatisfactory state of knowledge as to the pathology of gout the ocular conditions which arise can only be enumerated. Amongst them are eczema, tophi, hyperæmia, and œdema of the lids (Hirsch, Trousseau), conjunctivitis (Wagenmann, Leber, Trousseau, Hirsch), episcleritis, especially episcleritis periodica fugax (v. Vol. I, p. 271), and scleritis (Hutchinson, Galezowski, Hirsch, Pflüger, Fuchs, Wagenmann), sclerosing keratitis (Hirsch), band-shaped opacity (Galezowski, Hutchinson), iritis, cyclitis, and choroiditis (Hutchinson, Noyes, Boucheron, Wagenmann), punctate opacities in the lens (Bergmeister), recurrent vitreous hæmorrhages (Leber, Galezowski), with detachment of retina (Wagenmann), or retinitis proliferans (Bergmeister), retinal hæmorrhages (Hutchinson, Patterson, Gowers, Bull. Wagenmann), retinitis punctata albescens (Mooren, Hirschberg), papillitis and retrobulbar optic neuritis (Hutchinson, Gowers, Bull, Angelucci, La Torre).

HUTCHINSON.—R. L. O. H. Rep., vii, 1872; Brit. Med. Jl., 1872; Lancet, 1873, 1874; Med. Times and Gaz., 1882; Brit. Med. Jl., 1884. NOYES.—T. Am. O. S., 1873. LEBER.—

B. d. o. G., 1879. Boucheron.—Ann. d'Oc., xcvii, 1887. Brailey.—T. O. S., x, 1890.
Marcus Gunn.—v. Helmholtz'sche Festschrift, 1891. Bergmeister.—Wiener med. Woch.,
1894. Patterson.—Ann. of O., v, 1896. Trousseau.—Rec. d'O., 1896; A. d'O., xxi, 1901.
Wagenmann.—B. d. o. G., 1896; A. f. O., xliii, 1, 1897. Bull.—Med. News, 1897. La
Torre.—Arch. di Ott., v, 1898. Angelucci.—Rev. gén. d'O., 1899. Hirsch.—Vossius'
Sammlung, iii, 1899.

Rheumatism.—Acute rheumatism is doubtless an infectious disease ;
whether the so-called *Diplococcus rheumaticus* is a specific organism
or, as is more probable, a member of the group of streptococci is
as yet uncertain.   Iritis (Higgens, Laqueur), iridochoroiditis (B.
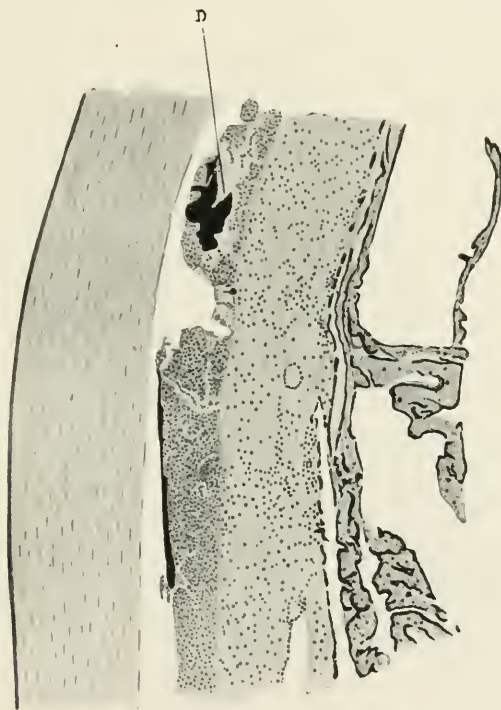
FIG. 841.—EXPERIMENTAL IRITIS.

Poynton and Paine, T. O. S., xxiii. Experimental iritis produced by intra-
venous inoculation of a rabbit with *Diplococcus rheumaticus*. The black mass (D)
shows diplococci on anterior surface of iris.

Schmidt), and cyclitis (Hugo Müller), are described as complications,
but the causal relationship of acute rheumatism to iritis is far from
proved.   Poynton and Paine obtained acute iritis in a few rabbits
after intra-venous injection of cultures of the *Diplococcus rheuma-
ticus*.   This iritis differed essentially from the iritis usually described
as "rheumatic iritis"; it was probably a septic iritis such as is not
infrequent after injection of virulent pyogenic organisms.   Episcleritis
and Tenonitis have been described during an acute attack, and there
are several cases of interstitial keratitis (Watson, Leber, Terrier,

v. Hippel, Albrand).   Uhthoff found in 221 cases of optic neuritis 4 due to rheumatism, and Königshöfer and Bruckner have reported cases.   Woinow reported a case of retro-bulbar neuritis, which has indeed often been attributed to this disease.   Embolism of the central artery may be due to the cardiac complications of acute rheumatism, though less frequently than the designation of the condition would lead one to expect (v. p. 1256).

Chronic muscular rheumatism is a much more frequent precursor of ocular disease, but it is difficult to decide how much must be attributed to gout in these cases.   Non-syphilitic iritis is commonly
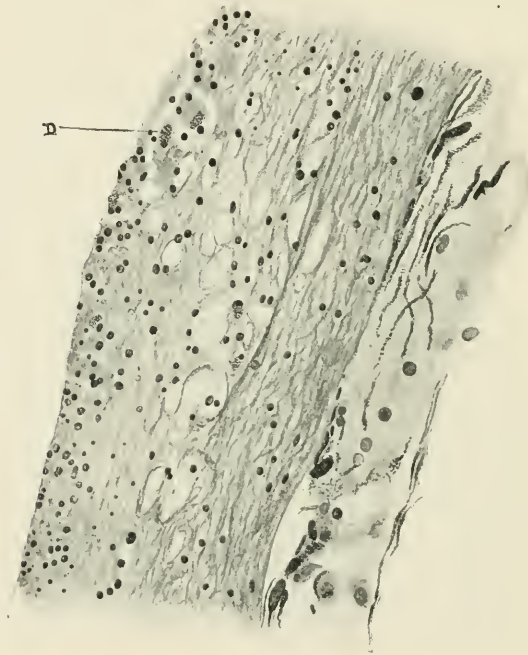
FIG. 842.—EXPERIMENTAL IRITIS.
From the same specimen. Showing diplococci in exudation more highly magnified.

called "rheumatic."   Nettleship in 71 cases of iritis found 30 (42 per cent.) due to syphilis and 23 (33 per cent.) in persons who suffered or whose near relatives suffered from rheumatism or arthritis.   Of these 23, 6 were attributable to gout and 1 to gonorrhœa, so that only 16 (23 per cent.) could be assigned to rheumatism.   Careful investigation will often reveal a history of gonorrhœa in these cases, and it is justifiable to have some scepticism as to the existence of rheumatic iritis.   In Nettleship's 16 cases only 2 were bilateral, whilst Schmidt-Rimpler holds that rheumatic iritis is generally bilateral.   Cyclitis (Boucheron) and disseminated choroiditis (Converse) have been attri-

buted to the same cause. Cases of interstitial keratitis have been recorded (Leber, Greeff). Albrand in 123 cases of interstitial keratitis found 12 with acute and 18 with chronic rheumatism. Episcleritis and Tenonitis, especially the former, show a great tendency to occur in "rheumatic" subjects. Uhthoff found 10 cases of optic neurtritis due to the chronic form in 221 cases. Retro-bulbar neuritis, again, is commonly attributed to chronic rheumatism in all obscure cases.

So far as evidence derived from the eye is concerned, I am inclined to regard "rheumatism" as an infectious disease of bacterial origin, and its ocular manifestations as due to endogenous infection (v. Chap. XXIV, etc.).

SPENCER WATSON.—Brit. Med. Jl., 1870. HUTCHINSON.—R. L. O. H. Rep., vii, viii, 1872; Brit. Med. Jl., 1872. SCHMIDT-RIMPLER.—A. f. O., xviii, 1, 1872. HUGO MÜLLER.—Dissertation, Greifswald, 1873. NOYES.—T. Am. O. S., 1873. HIGGENS.—Guy's Hosp. Rep., xix, 1874. LAQUEUR.—In Nagel's Jahresbericht, 1874. NETTLESHIP.—Brit. Med. Jl., 1876. LEBER.—B. d. o. G., 1879. TERRIER.—A. d'O., iv, 1884. BOUCHERON.—Ann. d'Oc., xcvii, 1887. MACNAMARA.—Westminster Hosp. Rep., 1889. ALBRAND.—Deutsche med. Woch., 1895. E. v. HIPPEL.—A. f. O., xlii, 2, 1896. KÖNIGSHÖFER.—Würtenberger med. Correspondenzbl., 1896. GREEFF.—Vossius' Sammlung, i, 1897. CONVERSE.—Ophth. Rec., 1900. POYNTON AND PAINE.—T. O. S., xxiii, 1903. CONNOR.—Ophth. Rec., 1905. BRÜCKNER.—A. f. A., lii, 1905. HOLMES SPICER.—Ophth. Rev., xxiv, 1905.

Myxœdema.—In addition to the changes in the skin of the lids (Chapman), loss of the eyebrows, tremor of the orbicularis palpebrarum (Landau), other ocular disorders have been noted, though the ætiological relationship is by no means certain. Such are interstitial keratitis (Grandclément, Treacher Collins)—interesting in connection with experimental results in dogs (v. Vol. I, p. 194)—optic neuritis (Bolte), optic atrophy (Wadsworth), neuroretinitis (Wagner), cataract (Callan), diplopia (Warfvinge), bitemporal hemianopia (Sanesi, Uhthoff). Thyroid feeding for this disease may, in excess, give rise to exophthalmos (Béclère, Lawford, Cunningham, Edmunds), and thyroidin amblyopia (Coppez, Birch-Hirschfeld and Inouye). In the latter there is chromatolysis of the retinal ganglion cells with secondary degeneration of the optic nerve-fibres.

WADSWORTH.—T. Am. O. S., 1884. LANDAU.—Berliner klin. Woch., 1887. LANDESBERG.—C. f. A., xii, 1888. NIXON.—Dublin Jl. of Med. Sc., 1889. BÉCLÈRE.—Gaz. des Hôp., 1894. CALLAN.—T, Am. O. S., 1895. UHTHOFF.—Berliner klin. Woch., 1897. CUNNINGHAM.—Jl. of Exp. Med., 1898. CHAPMAN.—Lancet, 1899. GRANDCLÉMENT.—Ann. d'Oc., cxxii, 1899. EDMUNDS.—T. O. S., xx, 1900. COPPEZ.—A. d'O., xx, 1900. WAGNER.—K. M. f. A., xxxviii, 1900. BOLTE.—Charité Annalen, xxviii, 1904. BIRCH-HIRSCHFELD AND INOUYE.—A. f. O., xli, 1905. TREACHER COLLINS.—T. O. S., xxvii, 1907. GOURFEIN-WELT.—A. d'O., xxvii, 1907.

DISEASES OF THE BLOOD.

Anæmia and chlorosis.—Apart from the subjective ocular symptoms so often present in simple anæmia and chlorosis changes not infrequently occur in the fundus. Raehlmann in eighty-six cases of general anæmia found definite retinal anæmia in 20 per cent., normal fulness of the vessels in 20 per cent., and marked dilatation in 60 per cent. In the last group the blood in the veins is lighter in colour than normal, and the reflex streak is more conspicuous. Gowers attributes

the broadening of the veins to diminished distension and to the flatten-
ing under the intra-ocular pressure. The brighter and broader reflex
streak is no proof of greater intra-vascular tension. Schmall found
the vessels normal in 20 per cent. of all chlorotics ; in 80 per cent. the
vessels looked pale and narrow, but the veins are often twice or thrice
as broad as the arteries. Schmall records tortuosity of the arteries as
a feature of chlorosis.

Pulsation of the arteries is common (Becker, v. Noorden), and is
usually only a locomotion pulse (v. p. 1254) (Schmall, Friedrichson).
Raehlmann divides the cases into two groups : those with arterial pulse
and hyperæmia, and those with retinal anæmia and no pulsation. The
former are plethoric individuals and the pulsation is attributed to
hydræmia. According to Thoma the vessel walls are more extensile in
anæmia, thus facilitating distension and tortuosity. Schmall attributes
the arterial pulse to the short, sharp cardiac pulse wave with diminished
general blood-pressure and diminished tension of the arterial walls.
Progressive peripheral centripetal venous pulse has been observed
(v. d. Osten-Sacken).

Retinal hæmorrhages have been described (Elschnig), but it is
doubtful if the cases were simple anæmias. Knies records the typical
appearances of albuminuric retinitis in one eye only, lasting six months
and disappearing entirely when the general condition improved (cf.
Gowers, Werner).

Optic neuritis and neuroretinitis have been frequently recorded
(Bitsch, Dieballa, Oliver, Schmidt, Bannister, Riegel, Engelhardt,
Uhthoff, Westcott and Pusey, Hawthorne, Pick). The diagnosis
must in many cases be accepted with reserve. Some are undoubtedly
pseudopapillitis (v. p. 933), occurring generally in hypermetropic eyes
and persisting indefinitely without disturbance of vision. In some
cases hæmorrhage into the nerve sheath affords the most reasonable
explanation, e. g. Litten and Hirschberg, where a central scotoma
gradually disappeared. The anæmia associated so frequently with
intra-cranial tumour must not be overlooked in this connection,
especially where bilateral choked disc is indisputable ; the syndrome
—headaches, vomiting, and optic neuritis—may be very misleading
if localising symptoms are absent or slight. Thrombosis of the
cavernous sinus as an explanation of optic neuritis and atrophy
(Hawthorne) is in most cases highly improbable. Thrombosis of the
central vein (q. v.) has been ascribed to chlorosis (Ballaban).

Scleritis, chronic iridocyclitis, rapid increase of myopia, etc., occur
in anæmic individuals, but the ætiological relationship is unproved.

Diminished coagulability is probably responsible for recurrent
spontaneous vitreous (v. p. 1256) and orbital (Wagner, Brunetière)
hæmorrhages.

BITSCH.—K. M. f. A., xvii, 1879. BECKER.—K. M. f. A., xviii, 1880. GOWERS.—Brit.
Med. Jl., 1881 ; The Ophthalmoscope in Medicine, London, 1904. EALES.—Ophth. Rev., iii,
1884. WILLIAMS.—Brit. Med. Jl., 1881. LITTEN AND HIRSCHBERG.—Berliner klin. Woch.,
1885. STEPHEN MACKENZIE.—Brit. Med. Jl., 1885. RAEHLMANN.—Virchow's Archiv, cii,
1885 ; K. M. f. A., xxvii, 1889. EDISON AND TEALE.—Brit. Med. Jl., 1888. FRIEDRICHSON.—
Dissertation Dorpat, 1888 ; A. f. O., xxxiv, 3, 1888. SCHMALL.—A. f. O., xxxiv, 1, 1888 ;
xxxv, 3, 1889. THOMA.—A. f. O., xxxv, 2, 1889. V. D. OSTEN-SACKEN.—Dissertation,

Dorpat, 1890. Beaumont.—T. O. S., x, 1890. Zumft.—Dissertation, Dorpat, 1891. Dieballa.—Deutsche med. Woch., 1896. v. Noorden.—In Nothnagel's Spec. Path., Wien, 1897. Holmes Spicer.—T. O. S., xvi, 1896. Schmidt.—A. f. A., xxxiv, 1897. Oliver.— T. Am. O. S., 1897. Bannister, Patrick.—Jl. of Nervous and Mental Diseases, xxv, 1898. Riegel.—Münchener med. Woch., 1899. Ballaban.—A. f. A., xli, 1900. Engelhardt.— Münchener med. Woch., 1900. Uhthoff.—Internat. Med. Congress, Paris, 1900. Pick.— Z. f. A., xi, 1904. Westcott and Pusey.—A. of O., xxxi, 1902. Hawthorne.—Brit. Med. Jl., 1902. Elschnig.—Wiener med. Woch., 1903. Werner.—T. O. S., xxv, 1905. Kampher- stein.—K. M. f. A., xliii, 1905. Pagenstecher.—A. f. A., lii, 1905. Wagner.—C. f. A., xxx, 1906. Brunetière.—Ann. d'Oc., cxxxv, 1906. Stevens.—Ophthalmology, 1906.

Secondary anæmias.—The anæmia of carcinoma of the stomach may be associated with hæmorrhages into the retina (Stephen Mackenzie, Buchwald) and white spots in the retina (retinitis cachechticorum, Pick). Pick found the changes in 30 per cent. of patients suffering from the cachexia of carcinoma of the stomach, and also in severe cases of cirrhosis, cancer, or syphilis of the liver, gastric and intestinal ulcers, tubercle of the lungs, intestines (Leonhardi-Aster) or peritoneum, etc. Retinal hæmorrhages in cases of severe burns are probably due to changes in the blood (Wagenmann). Similar conditions may be induced by the loss of blood from intestinal parasites, especially *Ankylostomum duodenale* (Rampoldi, Fischer, Natanson, Nieden) and *Botriocephalus latus* (Tschemolossow). The changes found in these secondary anæmias much resemble those of pernicious anæmia (q. v.), which should really be included in this group.

Hirschberg.—B. d. o. G., 1877. Leonhardi-Aster.—Deutsche Z. f. prakt. Med., 1878. Wagner.—Berliner klin. Woch., 1879. Rampoldi.—Ann. di Ott., ix, 1880; xvii, 1888. Stephen Mackenzie.—Lancet, 1883. Wagenmann.—A. f. O., xxxiv, 2, 1888. Fischer.— B. d. o. G., 1892; C. f. A., xx, 1896. Natanson.—In Nagel's Jahresbericht, 1894. Tschemolossow.—St. Petersb. med. Woch., 1894. Nieden.—Wiener med. Presse, 1897. Oliver.—T. Am. O. S., 1897. Pick.—K. M. f. A., xxxix, 1901.

Pernicious anæmia.—Biermer (1868), who first described pernicious anæmia, noticed retinal hæmorrhages, and the observation has been repeatedly confirmed : in fact they are seldom absent (Biermer, Horner). Bramwell found them in seven out of eight cases, Müller and Quincke in all cases at the height of the disease. They are there- fore of considerable diagnostic importance, for retinal hæmorrhages are rare in simple anæmia. The hæmorrhages occur in all parts of the fundus and at different depths in the retina, but most are flame-shaped and superficial. The centre of the spot is often paler than the edge, so that whitish spots with a red ring around them are very characteristic. White spots without extravasation of blood are common, and the typical picture of albuminuric retinitis, with a star at the macula, may be simulated. The edges of the disc are not infrequently blurred, and the papilla is usually pale and the vessels small. Sargent reported retinal hæmorrhages and detachment of the retina in a case.

Anatomical investigations have been carried out by a number of observers. The white centres of the hæmorrhages have been attributed to different causes. Manz found a conglomeration of white corpuscles with a definite limiting membrane, which he regarded as an ampulliform dilatation of a vessel's wall or of the lymph sheath (Eichorst, Nykamp). Bettmann found masses of lymphoid cells in various stages of degeneration ; in other cases only "varicose nerve-fibres" (cytoid

bodies, v. p. 1298) were present. Litten found sometimes only groups of white corpuscles; cytoid bodies may be present, the corpuscles having been absorbed to a greater or less extent. Sometimes only homogeneous masses of exudate are present. Uhthoff and Bondi found cytoid bodies with or without hæmorrhages. Sgrosso rightly regards the " varicose nerve-fibres " of other authors as altered white corpuscles (cytoid bodies). Homogeneous or finely granular deposits with a few " Körnchen " cells occur in the internuclear layer (Uhthoff). The retina is usually œdematous (Bettmann, de Schweinitz), and cystoid degeneration occurs. Litten described a case with definite retinitis— perivascular mantles of white corpuscles and diffuse cellular infiltration —but true inflammatory changes are generally absent. Manz attributes the hæmorrhages to capillary aneurysms, Litten to diapedesis, Nykamp and Sgrosso to rupture of the walls of the vessels. Fatty degeneration and other changes in the vessel walls, capillary emboli, etc., occur (Sgrosso). Changes occur in the extrinsic muscles, etc., as in other parts of the body (Fränkel, Rampoldi).

Biermer.—Correspondenzbl. f. Schweizer Aerzte, 1872. Horner.—K. M. f. A., xii, 1874. Manz.—C. f. d. med. Wissenschaft, 1875. Quincke.—Volkmann's Sammlung, 1876; Deutsches A. f. klin. Med., 1877. Bramwell.—Edin. Med. Jl., 1877. Fränkel.— Deutsches A. f. klin. Med., 1877. Litten.—Berliner klin. Woch., 1877, 1881. H. Müller.— Die prog. pern. Anämie, Zürich, 1877. Nykamp.—Berliner klin. Woch., 1877. Eichorst.— Die prog. pern. Anämie, Leipzig, 1878. Stephen Mackenzie.—Lancet, 1878. Uhthoff.— K. M. f. A., xviii, 1880. Weigert.—Virchow's Archiv, lxxix, 1880. Bettmann.—A. f. A., xi, 1881. Saundby and Eales.—Ophth. Rev., i, 1882. Carrington.—Lancet, 1883. Ransome and Mules.—Brit. Med. Jl., 1883. Kjellberg.—A. f. Kinderhk., 1884. Sargent.—A. of O., xxi, 1892. Bondi.—A. f. A., xxxiii, Ergänzungsheft, 1896. de Schweinitz.—T. Am. O. S., 1896. Sgrosso.—Lav. d. Clin. ocul. d. R. Univ. di Napoli, 1898. Pick.—K. M. f. A., xxxix, 1901. Fejér.—C. f. A., xxix, 1905.

Loss of blood.—Amblyopia or amaurosis following great loss of blood is not uncommon. Fries collected 106 cases from the literature up to 1876. Of these 36 per cent. were due to bleeding from the alimentary canal, 25 per cent. from the uterus, 25 per cent. artificially, generally by venesection, 7 per cent. from the nose, 5 per cent. from wounds, 1 per cent. from the lungs, 1 per cent. from the urethra. Increasing failure of vision may be due to repeated hæmorrhages (Hutchinson). The patients are always previously debilitated, generally over 40 years of age (youngest 2, oldest 77). In hæmatemesis men are twice as often affected as women (Pergens). The visual defect is bilateral; rarely unilateral (10—15 per cent.): in two thirds of the cases the defect is complete, in one fourth merely amblyopia. In 25 per cent. of the cases the defect commences during or immediately after the loss of blood, in 20 per cent. within 12 hours, in more than half later, generally 3—6 days, sometimes 18—21 days. In hæmatemesis, amongst 43 patients vision failed at once in 21 per cent., within 24 hours in 9 per cent., in 2—7 days in 51 per cent., in 7—14 days in 14 per cent., in 14—21 days in 5 per cent. (Pergens). Vision may return in a quarter of an hour, generally only after several days, weeks or months. In nearly half the cases vision does not improve. In hæmatemesis Pergens found 6 per cent. deaths, 36 per cent. bilateral blindness, 18 per cent. unilateral blindness, 18 per cent. great bilateral defect, 14 per cent. fairly good vision, 8 per cent. complete restoration.

Ophthalmoscopic examination soon after the hæmorrhage (Horstmann, Hirschberg, Schweigger, Litten) shows blurring of the disc, œdema of the retina diminishing towards the periphery, a few hæmorrhages and small white spots. Considerable changes may be present without failure of vision (Foerster). In many cases there is no change in the fundus (Leber). In unfavourable cases optic atrophy follows (v. Knies, Samelsohn, Hirschberg, Chevallereau, Snell). As in other diseases the appearance of the disc is little indication of the loss of vision. Defects in the field of vision (Horstmann, Uhthoff, Mandelstamm, Schmidt-Rimpler), light sense (Pergens), retrobulbar neuritis (Uhthoff, Borsch), etc., are reported. Cases of vitreous hæmorrhage and hyphæma (Beaumont), cataract (Pihl), night-blindness (Ancke), etc., belong to another category, not true complications but intercurrent disorders.

Anatomical observations have been published. Hirschberg found complete optic atrophy $3\frac{1}{2}$ years after the hæmorrhage, Ziegler ischæmic degeneration of the nerve, especially marked at the lamina cribrosa, Raehlmann changes in the blood-vessels. The clinical and anatomical observations point to the retina and optic nerve as the seats of the mischief. v. Graefe and Leber consider these due to hæmorrhage into the nerve sheath, Horstmann and Hirschberg to papillitis. Samelsohn considers that cerebral anæmia leads to lymph stasis inside the cranium; when the anæmia passes off the lymph is forced into the intervaginal space. In some cases there is disease (Samelsohn) or œdema (Schmidt-Rimpler) of the visual centres. Fries rightly discriminates between the early and late loss of sight. Ulrich lays stress upon retinal venous stasis, and Theobald regards thrombosis of the central vein as an explanation, but this can be true only of very exceptional cases. Ward Holden investigated the question experimentally on dogs.. He found changes in the ganglion cells of the retina and œdema of the retina. The most probable explanation of most cases is degeneration of the ganglion cells of the retina with secondary atrophy of the optic nerve, brought about by the anæmia and disordered nutrition (*vide infra*, "Toxic Amblyopias").

v. GRAEFE.—A. f. O., vi, 1, 1860. SAMELSOHN.—A. f. O., xviii, 2, 1872; xxi, 1, 1875. HUTCHINSON.—R. L. O. H. Rep., viii, 1874. FRIES.—K. M. f. A., xiv, 1876; xvi, 1878. HIRSCHBERG.—B. d. o. G., 1877; 1881; C. f. A., xvi, 1892. LANDESBERG.—K. M. f. A., xv, 1877. LEBER.—In G.-S., v, 1878. HORSTMANN.—K. M. f. A., xvi, 1878; Z. f. klin. Med., 1882. v. KRIES.—A. f. O., xxiv, 1, 1878. MANDELSTAMM.—C. f. A., iii, 1879. LITTEN.— Berliner klin. Woch., 1880. UHTHOFF.—A. f. O., xxvi, 1, 1880; xxxiii, 1, 1887. ULRICH.— A. f. O., xxvi, 3, 1880; K. M. f. A., xxi, 1883; A. f. O., xxxiii, 2, 1887. ANCKE.—C. f. A., x, 1886. SCHMIDT-RIMPLER.—K. M. f. A., xxv, 1887. ZIEGLER.—Beiträge, ii, 1887. GESSNER.—A. f. A., xix, 1889. RAEHLMANN.—Fortschr. d. Med., vii, 1889. BEAUMONT.—Ophth. Rev., xi, 1892. SOMYA.—C. f. A., xvi, 1892. PERGENS.—Ann. d'Oc., cxv, 1896. BORSCH.—Ann. d'Oc., cxix, 1898. BERTRAM.—Z. f. A., ii, 1899. HOLDEN.—A. f. A., xl, 1899. THEOBALD.—Am. Jl. of O., 1899. SWEET.—Ophth. Rec., 1899. PIHL.—C. f. A., xxiv, 1900. CHEVALLEREAU.—A. d'O., xxiii, 1903. DEPÈNE.—Allg. Med. Central Zeitung, 1903. SNELL.—T. O. S., xxiv, 1904. CARLINI.—La Clinica oculistica, 1906.

Leukæmia.—The ophthalmoscopic changes found in leukæmia were first described by Liebreich, but the picture in his 'Atlas' is not the most typical. The fundus is orange coloured, the veins are dilated and tortuous, often with white lines along them, and are bright red, not

dark; the arteries are small and pale yellowish red. The retina is often hazy and œdematous, the disc pale, seldom swollen (Oeller). Retinal hæmorrhages and small white spots are common. The peculiar colour of the fundus may be absent, the picture resembling that of chlorosis. Schirmer found retinitis in one case out of five leukæmic patients. The changes may be overlooked, for Leber found them specially between the equator and the ora serrata. Similar fundus conditions are found in acute leukæmia.

Anatomical observations show the conspicuous excess of leucocytes in the blood which is characteristic of the disease. The tissues are widely infiltrated with white corpuscles, and I have found that this feature is particularly marked in acute leukæmia (v. Fig. 843). The nature of the white spots and patches, which sometimes attain considerable size, varies in different cases. v. Recklinghausen found

FIG. 843.—ACUTE LEUKÆMIA.
Photograph by Coats. Showing infiltration and hæmorrhages in the retina, and dense infiltration of the choroid with leucocytes.

"varicose nerve-fibres" (cytoid bodies, v. p. 1298), Perrin corpuscles with fatty granules (Fettkörnchenzellen) in the outer layers of the retina, Leber extravasations of blood with great excess of white corpuscles. Deutschmann and Murakami vascular ectasiæ filled with leucocytes. Spots composed of white corpuscles surrounded by a ring of red corpuscles occur as in pernicious anæmia (q. v.) (Leber, Deutschmann). The white lines along the vessels are explained by the mantle of leucocytes often found distending the perivascular spaces. The papilla is infiltrated and hæmorrhages are found in all the layers of the retina and even in the choroid. Schmidt-Rimpler has noticed extravasations of red corpuscles whilst the neighbouring vessels were packed with white corpuscles. v. Michel attributes the hæmorrhages to venous thrombosis. The choroid, apart from hæmorrhages, may be much thickened by infiltration with lymph cells (Roth, Oeller,

Osterwald, Bäck); this accounts for the orange fundus reflex. Aggregations of lymphoid cells are found in the ciliary body and sclerotic (Bäck, Kerschbaumer). Kerschbaumer describes a case in which Tenon's capsule and the inter-vaginal space of the optic nerve were packed with leucocytes. The exact mechanism of the production of the white masses in the retina has been a subject of dispute and cannot be settled definitely. They may be due to actual rupture of the vessels (Roth), diapedesis (Leber), stasis, diapedesis, and multiplication of leucocytes (Murakami), vascular ectasiæ with subsequent degeneration of the vessel walls (Murakami), etc.

Besides retinitis other changes may occur in leukæmic eyes, e.g. hyphæma (Sorger), vitreous hæmorrhage and secondary glaucoma (Saemisch), vitreous opacities (Schmidt-Rimpler), detachment of the retina by hæmorrhage (Scholz), choked disc (Kerschbaumer), retrobulbar neuritis (Schmidt-Rimpler), iritis (v. Michel), iridocyclitis (Horner), etc.

Lymphomata of the lids and orbit sometimes occur in leukæmia and pseudo-leukæmia (v. Vol. I, p. 19).

LIEBREICH.—Deutsche Klinik, 1861; Atlas, Pl. x, Fig. 3, Berlin, 1863. BECKER.—A. f. A., i, 1869. LEBER.—K. M. f. A., vii, 1869; in G.-S., v, 1877; A. f. O., xxiv, i, 1878. SAEMISCH.—K. M. f. A., vii, 1869. ROTH.—Virchow's Archiv, xlix, 1870. DEUTSCHMANN.—K. M. f. A., xvi, 1878; B. z. A., iv, 1892. v. MICHEL.—Deutsches A. f. klin. Med., xxii, 1878; A. f. O., xxvii, 2, 1881. OELLER.—A. f. O., xxiv, 3, 1878. FRIEDLÄNDER.—Virchow's Archiv, lxxviii, 1879. OSTERWALD.—A. f. O., xxvii, 3, 1881. JESSOP.—T. O. S., vi, 1886. HIRSCHBERG.—C. f. A., xi, 1887. EDMUNDS.—T. O. S., x, 1890. HARTRIDGE.—T. O. S., xiii, 1893. KERSCHBAUMER.—A. f. O., xli, 3, 1895. DUCLOS.—Ann. d'Oc., cxvii, 1897. FINLAYSON.—Brit. Med. Jl., 1898. FRÄNKEL.—Münchener med. Woch., 1898. PUCCIONI.—Ann. di Ott., xxvii, 1898. SORGER.—Münchener med. Woch., 1898. BÄCK.—Z. f. A., i, 1899. ELSCHNIG.—Wiener med. Presse, 1899. SCHOLZ.—Ungarische B. z. A., 1899. FIELCHENFELD.—A. f. A., xli, 1900. HOCHHEIM.—A. f. O., li, 1900. GRUNERT.—C. f. A., xxv, 1901. MURAKAMI.—K. M. f. A., xxxix, 1901. GLINSKI.—Virchow's Archiv, clxxi, 1903. ORLOW.—In Nagel's Jahresbericht, 1903. MELLER.—Z. f. A., xiv, 1905. STOCK.—K. M. f. A., xliv, 1906.

Polycythæmia.—In polycythæmia, which is characterised by great increase in the number of red corpuscles, Uhthoff has found distension of the retinal veins and dark coloration of the blood seen ophthalmoscopically.

UHTHOFF.—K. M. f. A., xliv, 1906.

Scurvy, purpura, hæmophilia.—Hæmorrhages in and about the eye are not uncommon in these diseases. In scurvy $3\frac{1}{2}$ per cent. have ocular affections (Fialkowsky), including interstitial keratitis, corneal ulcer (Ewmeniow), hyphæma (Adler), retinal hæmorrhages (Wegscheider, Hale White, Belawsky), neuroretinitis (Stephen Mackenzie), choked disc (Seggel), orbital hæmorrhage (Krückow, Magnus, Heubner, Franke), xerosis and night-blindness, etc. Sulzer's case of spontaneous conjunctival hæmorrhage is said not to be due to hæmophilia.

WEGSCHEIDER.—Deutsche med. Woch., 1877. MAGNUS.—Deutsche med. Woch., 1878. FIALKOWSKY.—C. f. A., iv, 1880. STEPHEN MACKENZIE.—T. O. S., i, 1881. HALE WHITE.—Lancet, 1883. DENIG.—Münchener med. Woch., 1895. EWMENIOW, WASKRESENSKY.—A. f. A., xxxiv, 1896. GOH.—A. f. O., xliii, 1, 1897. SEGGEL.—K. M. f. A., xxxvii, 1899. HEUBNER.—Berliner klin. Woch., 1903. WEIL.—Z. f. A., ix, 1903. HIRSCHBERG.—C. f. A., xxvii, 1903. AXENFELD.—Münchener med. Woch., 1904. FRANKE.—Münch. med. Woch., 1904. STILL.—Brit. Med. Jl., 1906. SULZER.—Ann. d Oc., cxxxvii, 1907.

In purpura (morbus maculosus Werlhofii) retinal hæmorrhages (Stephen Mackenzie, Goodhart), retinal, choroidal, and scleral hæmorrhages (Ruc), optic neuritis (Lawford, Nettleship) are recorded.

Ruc.—Union méd., 1870. Stephen Mackenzie.—Med. Times and Gaz., 1877; Brit. Med. Jl., 1882. Goodhart.—Lancet, 1878. Lawford, Nettleship.—T. O. S., ii, 1882. Spiro.—C. f. A., xxii, 1898. *Marx.—A. f. O., lxiv, 1906. Schultz-Zehden.—K. M. f. A., xlv, 1907.

Retinal hæmorrhages (Galezowski, Hirschberg, Bramwell, Schnaudigel), vitreous hæmorrhage (Wagenmann, Weber, Hauenschild, Uhthoff), orbital hæmorrhage (Priestley Smith, Valude, Hahn), bleeding from the conjunctiva (Grandidier, 5 in 334 cases, Schmidt-Rimpler, Müller, Shirley, Jessop and Stephenson) are recorded in hæmophilia.

Hirschberg.—A. f. A., viii, 1879. Bramwell.—Edin. Med. Jl., 1886. Schmidt-Rimpler.—K. M. f. A., xxv, 1887; Die Erkrankungen, etc., Wien, 1905. Priestley Smith.—R. L. O. H. Rep., xii, 1888. Shirley.—New York Med. Jl., 1891. Müller.—A. f. Gynäk., 1893. Jessop and Stephenson.—Ophth. Rev., xiv, 1895. Valude.—Ann. d'Oc., cxvii, 1897. Wagenmann, Weber.—A. f. O., xliv, 1, 1897. Schnaudigel.—A. f. O., xlvii, 3, 1899. Zirm.—C. f. A., xxiii, 1899. Hahn.—Dissertation, Tübingen, 1900. Hauenschild.—Münchener med. Woch., 1900.

Asthenia.

Keratomalacia.—The cornea is specially likely to be affected in asthenic conditions produced by any disease, probably owing to the absence of blood-vessels in it. In babies and young children corneal ulcers are liable to occur under these circumstances. Very characteristic are the central symmetrical chronic ulcers which show very slight infiltration and inflammatory reaction, undergo little or no vascularisation, and often end in the formation of a clear facet, probably owing to the absence of pabulum for the production of scar tissue. In the severer forms of malnutrition similar ulcers appear, less commonly symmetrical and centrally situated; they are characterised by localised necrosis, leading rapidly to perforation and the extrusion of a knuckle of iris. The aperture in the cornea is usually 3—4 mm in diameter; on account of its size and the extremely defective regenerative reaction excision of the prolapse is liable to be followed by shrinking of the globe. With restoration of normal nutrition the cicatrix usually becomes flat. In keratomalacia both corneæ undergo necrotic changes *en masse*. The centres first become grey from deep infiltration, which spreads until the whole corneæ are involved. The epithelium is desquamated, organisms invade the moribund or necrotic tissues, and in a short time perforation occurs. The whole cornea may be cast off, with extrusion of the lens and exposure of the vitreous. Panophthalmitis may ensue, but more commonly the child dies before this stage is reached. The conjunctiva is usually xerotic. Children under six months of age are most affected, and they generally suffer from uncontrollable diarrhœa; in many cases the disease is preceded by an exanthem or other debilitating disorder. The younger the child the worse as a rule is the prognosis both for life and for the cornea. Many of the children suffer from congenital syphilis but the disease is not essentially syphilitic.

The affection of the cornea was early regarded as secondary to an encephalitis (v. Graefe, Klebs), but this view has been abandoned (Jastrowitz, Foerster). There can be no doubt that the corneal condition is primarily a necrosis (Foerster, Elschnig), and this has been attributed to thrombosis of the pericorneal vessels, to drying of the cornea from defective closure of the lids, to loss of sensation as in neuroparalytic keratitis, etc. It is certain that it is not due to the xerosis bacillus, which is present in large numbers, but the invasion of the badly-nourished tissues by pathogenic organisms follows desquamation of the epithelium and is an important factor in the later stages. Staphylococci and streptococci are frequently present (Schanz, Uhthoff and Axenfeld. Leber and Wagenmann, Dötsch).

Lobo.—K. M. f. A., iv, 1866. v. Graefe.—A. f. O., xii, 2, 1866. Hirschberg.—Berliner klin. Woch., 1868. Jastrowitz.—A. f. Psychiatrie, ii, iii, 1870. Bezold.—Berliner klin. Woch., 1874. Foerster.—In G.-S., vii, 1877. Leber.—A. f. O., xxix, 3, 1883. Thalberg.—A. f. A., xii, 1883. Schleich.—Mitt. a. d. ophth. Klinik in Tübingen, 1884. Schulz.—A. f. O., xxx, 4, 1884. Weeks.—A. f. A., xvii, 1887 (Bibliography). Leber and Wagenmann.—A. f. O., xxxiv, 4, 1888. Schanz.—A. f. A., xxv, 1892. Zirm.—Wiener klin. Woch., 1895. Uhthoff and Axenfeld.—A. f. O., xlii, 1, 1896. Elschnig.—Wiener med. Woch., 1899. Dötsch.—A. f. O., xlix, 2, 1900. Baer.—K. M. f. A., xxxix, 1901.

Night=blindness and xerosis.—In patients who are old enough to be tested xerosis is frequently associated with night-blindness. This "acute idiopathic" night-blindness, which may occur independently of xerosis, is found in sailors, soldiers, peasants—especially in Russia, etc., during periods of religious fasting—and also sporadically in badly-nourished people. Uhthoff reports a case in a fanatic vegetarian, Foerster in nephritis, and others in diseases of the liver, malaria, miners' nystagmus, etc. In 500 alcoholics Uhthoff noted Bitot's spots, with or without night-blindness, in 5 per cent., and he considers that alcohol is in itself occasionally the cause. It is frequent in scurvy (v. Michel and others). In most cases malnutrition alone is insufficient to produce the condition, and exposure of the retinæ to bright light is essential. One eye may indeed be protected during the daytime and will be available for use at night whilst the uncovered eye becomes night-blind (Nettleship). Exposure to bright light, however, is not invariably necessary (Foerster, Krienes).

Bitot.—Gaz. hebd., 1863. Weiss.—Berliner klin. Woch., 1873. v. Michel.—Bayr. ärztl. Intelligenzbl., 1882. de Gouvea.—A. f. O., xxix, 1, 1883 (Bibliography). Kubli.—A. f. A., xvii, 1887. Uhthoff.—Berliner klin. Woch., 1890. *Krienes.—Ueber Hemeralopie, Wiesbaden, 1896 (Bibliography). Schmidt-Rimpler.—Art.-Hemeralopie in Eulenburg's Realencyclopädie, 1896. Trantas.—Rec. d'O., 1900. Parsons —Lancet, 1908.

Other ocular lesions described as associated with asthenia are optic neuritis (Immermann, Horschel), amblyopia (Jennings, Lopez), glaucoma (Laqueur, Mooren, Jacobson, Lange), cycloplegia (Gubler, Jacobson, Belawsky, Hutchinson, Landesberg, Mooren, Rampoldi), cataract (Hogg and others).

Horschel.—Deutsche med. Woch., 1882. Immermann.—A. f. Psych., xix, 1887. Jennings.—Am. Jl. of O., 1900. Lopez.—Ann. de Oft., 1900. Laqueur.—A. f. O., xxxvi, 2, 1880. Mooren.—A. f. A., xiii, 1884. Jacobson.—A. f. O., xxxii, 3, 1886. Lange.—Vossius' Sammlung, 1, 1896. Gubler.—Arch. gén. de Méd., 1860. Hutchinson.—Med. Times and Gaz., 1879. Rampoldi.—Ann.. di Ott., xi, 1882. Collins.—Lancet, 1886; R. L. O. H. Rep., xi, 1887.

Diseases of the Nasal Sinuses.

Reference has already been made to tumours and empyemata of the nasal sinuses (*v.* Vol. II, p. 747), and to rupture and compression of the optic nerve as the result of fracture of the skull or other traumatism (*v.* p. 1182). The anatomy of these parts has been described by Berger and Tyrmann, Ziem, Hajĕk, Killian, Avellis, Stanculéanu, Paunz, and Onodi. It may be noted that the wall of the optic foramen is sometimes as thin as paper (Berger and Tyrmann) and that there may be actual lacunæ in it (Gallemaerts, Holmes, Onodi, and others). Attention is here particularly directed to disease of the optic nerve, usually in the form of a retrobulbar neuritis, which may be due to disease of the sinuses other than that associated with injury. There can be little doubt that many obscure cases of retrobulbar neuritis are caused by such disease. Berger lays stress upon concentric diminution of the field of vision as a sign of implication of the optic nerve in the neighbourhood of the foramen optici. Birch-Hirschfeld, on the other hand, has shown that the papillomacular bundle is particularly vulnerable, and that the usual sign of retrobulbar neuritis—central scotoma—is the commonest symptom. Ophthalmoscopic signs are absent in the early stages (Berger, Bull, Axenfeld, Coppez, Risley, Oliver and Wood, Harlan, and others). Later papillitis (Würdemann, Villard, Lane, Ewetzky, Rohmer, and others), papillœdema (Ziem, Johnson), venous hyperæmia, etc., may be found. Birch-Hirschfeld has collected and collated the literature, adding four cases, one of which was examined anatomically.

Berger and Tyrmann.—Die Krankheiten der Keilbeinhöhle, etc., 1886. Ziem.—Monatsschrift f. Ohrenheilk., 1893. Hajek.—Path. u. Ther. d. entzündl. Erkrankungen d. Nebenhöhlen d. Nase, Leipzig, 1899. Stanculéanu.—A. d'O., xxii, 1902. Paunz.—A. f. A., lii, 1905. Onodi.—Z. f. A., xii, 1904. Bull.—Ophth. Rev., xviii, 1899; Med. Rec., 1899. Depage.—Rev. gén. d'O., 1902. Fish.—A. f. A., lii, 1905; Brit. Med. Jl., 1907. Harlan.—T. Am. O. S., 1900. Heilmaier.—Z. f. A., ii, 1899. Hoffmann.—Z. f. A., xvi, 1906. Holmes.—A. of O., xxv, 1897. Jessop.—T. O. S., xxiii, 1903. Johnston.—Ophth. Rec., 1905. Knapp.—A. of O., xxxii, 1903. Lane.—Brit. Med. Jl., 1893. de Lapersonne.—A. d'O., xviii, 1898. Mendel.—C. f. A., xxv, 1901. Miller.—Brit. Med. Jl., 1900. Nieden.—A. f. A., xvi, 1886. Oliver and Wood.—Am. Jl. of Med. Sc., 1902. Posey.—Ophth. Rec., 1902. Risley.—T. Am. O. S., 1900. Vieusse.—Rec. d'O., 1899. Villard.—A. d'O., xv, 1895. Vossius.—Z. f. A., iv, 1900. Würdemann.—Ophth. Rec., 1905. Alexander.—Münch. med. Woch., 1905. Glegg and Hay.—Lancet, 1905. Bellinzona, Bossalino, di Santo.—Ann. di Ott., xxxiv, 1905. *Birch-Hirschfeld.—A. f. O., lxv, 3, 1907 (Bibliography).

Infectious Diseases.

Measles.—The initial stage of measles is accompanied by conjunctivitis and lacrymation ; the conjunctival secretion may carry infection. Bacteriological examination has shown the presence of diplococci resembling pneumococci (Stschegolew), pneumococci (Hertel), streptococci (Schottelius), a short slender bacillus resembling the influenza bacillus (Giarré and Picchi), etc. Œdema of the lids, with or without exanthematous spots, occurs (*v.* Michel), and blepharitis often follows. Gangrene of the lids is recorded (Fieuzal, Knies, St. Martin). The conjunctivitis may be pseudomembranous (Hirschberg, Schmidt-Rimpler, Hertel), and may lead to loss of the eyes from corneal ulcera-

tion (Mason).   More frequent is phlyctenular ophthalmia, coming on usually after the subsidence of the rash.   Corneal ulcers are common, keratomalacia rare (Bezold, Fischer, Beyer, Trantas).   Trantas records superficial punctate keratitis.   Iritis occurs only secondarily to corneal complications.   Metastatic ophthalmia gives rise to one form of pseudoglioma (q. v.) (Stieren, Treacher Collins, and others).   Choroiditis is reported by Vossius, and myopia has been ascribed to posterior scleritis after measles (Jacobson, Müller).   Albuminuric retinitis (Horner), and retinitis of similar type without albuminuria (Sotow), are mentioned.   Optic neuritis is rare—9 out of 253 cases occurring in infectious diseases (Uhthoff) ;   Groenouw has collected 17 cases, once in an adult (Arago).   It occurs in or after the third week and is bilateral.   Optic atrophy (Nagel) or restoration of vision may follow. Meningitis may cause paralysis of extrinsic muscles (Wadsworth, Keller), etc., and is probably responsible for optic neuritis.   Amaurosis without ophthalmoscopic abnormalities is described (Nagel), probably uræmic in origin.   Acute dacryoadenitis (Lindner, Adler) and dacryo-cystitis occur.   Periostitis (Strubell) and bilateral orbital cellulitis (Gallemaerts) are recorded.   Measles may have a beneficial effect upon trachomatous pannus (Fialkowsky) and interstitial keratitis (Harlan). or it may lead to necrosis of a leucoma adherens (Hirschberg).

v. GRAEFE.—A. f. O., xii, 2, 1866.   HIRSCHBERG.—Berliner klin. Woch., 1869. MASON.—R. L. O. H. Rep., vii, 1871.   KÖNIGSTEIN.—Oesterreich. Jahrb. f. Pädiatrik, 1876. v. MICHEL.—In G.-S., iv., 1876.   WADSWORTH.—T. Am. O. S., 1880.   KELLER.—Monats-schrift f. Ohrenheilk., 1888.   STEPHENSON.—T. O. S., viii, 1888.   COGGIN.—Am. Jl. of O., 1890.   WOODS.—A. of O., xxi, 1892.   STRUBELL.—Münchener med. Woch., 1898. FLEMMING.—Brit. Med. Jl., 1899.   SOTOW.—Jahrb. f. Kinderheilk., 1900.   STIEREN.—Pennsylvania Med. Jl., 1900.   UHTHOFF.—B. d. o. G., 1900.   TRANTAS.— Die ophth. Klinik, 1901.   GIARRÉ AND PICCHI.—Die med. Woche, 1901.   HARLAN.—A. of O., xxx, 1901. HERTEL.—A. f. O., liii, 1902.   BONDI.—In Nagel's Jahresbericht. 1903.   SCHOTTELIUS.— K. M. f. A., xlii, 1904 ;   Münch. med. Woch., 1904.   AXENFELD.—Münchener med. Woch., 1904.   DE VAUCRESSON.—Ann. d'Oc., cxxxv, 1906.

Scarlet fever.—Lid affections, apart from œdema, which may be a sign of renal complication, are rare (abscess, Jackson ; gangrene, St. Martin).   Conjunctivitis is not an integral part of the clinical picture as in measles, but pseudo-membranous conjunctivitis may occur. gene-rally due to streptococci (Debièrre, Uhthoff, Stöwer), less frequently to the diphtheria bacillus.   Corneal ulcers are not common (c. f. Kendall). Mooren saw scarlet fever two days after a cataract operation with loss of the cornea and eye.   Keratomalacia may occur (v. Graefe).   Albu-minuric retinitis (q. v.), retinitis of the same type without albuminuria (Vance), embolism of the central artery (Hodges), are reported.   Optic neuritis is rare, 3 in 253 cases occurring in infectious diseases (Uhthoff). Groenouw collected 5 cases, 1 with albuminuria (Barlow), 3 without (Betke, Vance, Pflüger) ;   meningitis may be present (Thomas). Orbital cellulitis has been observed (Gregory, Deval) ;   Nettleship attributed a case of unilateral optic atrophy to this cause.   Strubell recorded periostitis.   Dacryocystitis (Kendall) and dacryoadenitis (Linder), as well as paralysis of ocular muscles (Lenhartz), are rare. Scarlet fever is a not uncommon cause of uræmic amaurosis (Ebert, Reimer, Power, Loeb, Foerster, Barlow, Becher).   Albuminuric

retinitis is rare in the acute nephritis of scarlet fever (Hutchinson, Marchand) but may result from the subsequent chronic nephritis (Aufrecht).

DEVAL.—Ann. d'Oc., xxi, 1849.   v. GRAEFE.—A. f. O., xii, 2, 1866.   MARTIN.— St. Bartholomew's Hosp. Rep., 1867.   EBERT.—K. M. f. A., vi, 1868.   BETKE.—K. M. f. A., vii, 1869.   HUTCHINSON.—Lancet, 1871.   POWER.—Practitioner, 1871.   FOERSTER.— K. M. f. A., x, 1872.   VANCE.—Philadelphia Med. and Surg. Rep., 1873.   BAYLEY, POTTER.—Lancet, 1877.   PFLÜGER.—A. f. O., xxiv, 2, 1878.   NETTLESHIP.—Med. Times and Gaz., 1880.   BARLOW.—Med. Times and Gaz., 1881.   KENDALL.—Brit. Med. Jl., 1883. MARCHAND.—Berliner klin. Woch., 1883.   HODGES.—Ophth. Rev., iv, 1885.   WEEKS.— C. f. A., ix, 1885.   AUFRECHT.—Deutsches A. f. klin. Med., 1888.   DEBIÈRRE.—Ann. d'Oc., cxi, 1894.   JACKSON, SIDNEY.—Brit. Med. Jl., 1895.   STRUBELL.—Münchener med. Woch., 1898.   STÖWER.—Z. f. A., ii, 1899.   SISSON.—Med. Fortnightly, 1904.   WERNER.— Ophthalmoscope, 1905.

Smallpox.—Blindness was frequently caused by smallpox (1·3— 2·5 per cent. [Hirschberg 9 per cent.] of cases) before the introduction of vaccination.   Intra-uterine infection from the mother may cause phthisis bulbi (Panas).   Of diseases of the eye caused by smallpox 38—78 per cent. are affections of the cornea (H. Cohn).   They never occur before the tenth day (Adler), generally from the twelfth to the fourteenth day (Makuna).   The eruption is common on the lids, causing œdema and secondary cellulitis of the face (Landesberg) ; distortion of the lids frequently follows.   There is generally conjunctivitis, sometimes with marked chemosis (Zülzer) or hæmorrhage (Gorkom).   Pustules may occur on the conjunctiva (Adler in 30 per cent., Hebra in 1 per cent., of cases) ; Wagenmann described a case in which the eruption was limited to the conjunctiva.   There has been much discussion as to whether the keratitis is due to pustules (Horner) ; it is now generally accepted that it is not.   Small circumscribed infiltrations (Hirschberg, Bergmeister, Adler), interstitial keratitis (Adler, Hirschberg, Bock, Coccius—16 times in 58 cases, Findlay), and hypopyon ulcers occur.   Hirschberg observed neuroparalytic keratitis, Manz and Adler keratomalacia.   The severity of the corneal complication is not always proportional to that of the general disease (Hackenberg, Bergmeister, Schmidt-Rimpler).   Primary iritis is not uncommon (Coccius 17 per cent., Adler 13 per cent. of all cases) ; it is generally part of a general uveitis with choroiditis and vitreous opacities, and may lead to complicated cataract (v. Graefe, Adler).   Simple plastic iritis and chronic iridocyclitis, however, also occur.   Retinitis (Manz), retinal hæmorrhages, albuminuric retinitis (Adler), retinal detachment (v. Hippel), uræmic amaurosis, etc., may occur.   Gowers and v. Hippel observed optic atrophy, Leber, Adler and others neuroretinitis, Prothon choked disc, Riedl retrobulbar neuritis.   Glaucoma is mentioned (Adler, Coccius, v. Graefe—hæmorrhagic glaucoma in hæmorrhagic smallpox, Watson).   Other complications are dacryocystitis (Gambarotto), lacrymal fistula (Mooren), dacryoadenitis, periostitis (Landesberg, Magnus), etc.

v. GRAEFE.—A. f. O., xv, 3, 1869.   COCCIUS.—De Morbis oculi etc., Leipzig, 1871. HIRSCHBERG.—Berliner klin. Woch., 1871.   HORNER.—Korrespondenzbl. f. Schweitzer Aerzte, 1871.   HULKE, HUTCHINSON.—Brit. Med. Jl., 1871.   NETTLESHIP.—R. L. O. H. Rep., vii, 1871.   WATSON.—Practitioner, 1871.   ADLER.—A. f. Derm. u. Syphilis, 1874 (Bibliography).   BERGMEISTER.—K. M. f. A., xii, 1874.   LANDESBERG.—Beitrag zur

variolösen Ophthalmie, Elberfeld, 1874. Makuna.—Brit. Med. Jl., 1882. Hutchinson, Jr.—
T. O. S., vi, 1886. Wagenmann.—A. f. O., xli, 1, 1895. Finlay.—A. f. A., xxxvii, 1898.
*Groenouw.—In G.-S., xi, 1, 1903 (Bibliography).

Vaccination.—Self-infection of the eyes from a vaccinated arm is
rare (8 in 50 cases, Pihl). In 3 cases physicians have vaccinated
themselves about the eyes accidentally (Critchett, Sénut, Eagleton).
Usually the patients are women who nurse vaccinated children. One
eye only is affected ; in 43 out of 47 cases the lids were inoculated, in
3 the conjunctiva, and in 1 the cornea (Schirmer). The incubation
period is 3—4 days, occurring usually 10—15 days after vaccination
of the child. Primary affection of the conjunctiva has been recorded
seldom (Schirmer, Purtscher, Eagleton, v. Forster, Pihl), though exten-
sion to the conjunctiva is not uncommon ; in 3 out of 5 cases corneal
complication arose, twice ulceration, once keratitis profunda. Inocula-
tion of one lid from the other by contact is not uncommon, so that
symmetrical affection of the upper and lower lids is seen. Primary
infection of the cornea is very rare, though two or three undoubted
cases are recorded (Critchett and others). In each case a physician has
accidentally inoculated himself either with the lancet or by a broken
vaccine tube. Secondary affection of the cornea is less uncommon ;
Schirmer collected 8 cases of ulceration and 5 of deep keratitis in 46
cases. Ulceration is usually sickle-shaped and marginal ; in one case a
large leucoma resulted (Schirmer), in another the eye was lost (Calhoun).
Keratitis profunda post-vaccinolosa has been specially studied by
Schirmer. It occurs late, when cicatrisation is going on in the lid. It
is characterised by deep infiltration with denser streaks running in
various directions. The infiltration is often disc-shaped. There is some
iritis, and precipitates (" k. p.") sometimes occur on the back of the
cornea. The condition is very chronic and leaves permanent haze. In
some cases it has been preceded by transitory central ulceration
(Schirmer, Schmitz).

Vetter.—A. f. O., vi, 2, 1860. Critchett.—Med. Examiner, 1876. Hirschberg.—
A. f. A., viii, 1879; C. f. A., ix, 1885; xvi, 1892. Knaggs.—T. O. S., i, 1881. Calhoun.—
Am. Med. Assoc., 1882. Sénut.—Rec. d'O., 1885. Berry.—Brit. Med. Jl., 1890. James.—
T. O. S., xi, 1891. Tatham Thompson.—T. O. S., xii, 1892. Zimmermann.—A. of O.,
xxi, 1892. Purtscher, Schapringer.—C. f. A., xix, 1895. Fröhlich.—A. f. A., xxxiii,
Ergänzungsheft, 1896. Eagleton.—Ophth. Rec., 1899. v. Forster.—Münchener med.
Woch., 1900. Schirmer.—Vossius' Sammlung, iii, 1900. Ormsby.—Lancet, 1901. Pihl.—
K. M. f. A., xxxviii, 1901. Pooley.—New Amsterdam Eye and Ear Hosp. Rep., 1904.

Chickenpox.—The skin of the lids may be affected by the eruption ;
sometimes it appears first in this situation (Comby). Gangrene of the
lids in a child æt. 8 months has been reported (Römer). The con-
junctiva is rarely affected by the eruption (Hilbert). Acute iritis
(Steffan), iridochoroiditis (Hutchinson), optic neuritis (Hutchinson)
occurring after varicella are probably not due to it.

Hutchinson.—R. L. O. H. Rep., vi, 1869. Hutchinson Jr.—Ophth. Rev., v, 1886.
Römer.—Vossius' Sammlung, iii, 1900. Hilbert.—C. f. A., xxvi, 1902.

Erysipelas.—Since erysipelas is common on the face the lids are
often affected, and gangrene has been observed (Lamzweede, 1656).
Abscesses, sometimes multiple (Elschnig), are not uncommon.

Secondary extension to the eye (Biermann) or orbit with atrophy of one (Rampoldi) or both optic nerves (v. Arlt) occurs. Chronic thickening of the lids may ensue (Smith, Lavraud, Anderson Critchett, and others). Bullous (Coursserant) or hypopyon keratitis (Schmidt-Rimpler) has been seen, also anterior staphyloma (Scimeni), and neuroparalytic keratitis (Neve). Dacryoadenitis (Carre, Lindner) and dacryocystitis (v. Knies) are recorded. It must be remembered that dacryocystitis and ophthalmic herpes are not infrequently confounded with true erysipelas. Orbital cellulitis is not uncommonly due to facial erysipelas; Schwendt found 13 cases in 44 of orbital cellulitis not due to trauma or extension from the orbital bones. It occurs 2—4 days after the outbreak of the erysipelas, rarely after disappearance of the latter (Poland, Parinaud). The disease may be bilateral, either by direct infection or by way of the cavernous sinus (Leber). Signs of retrobulbar neuritis (Leber, Pagenstecher) or optic neuritis (Lubinsky, Karafiath, Hallermann), or the picture of embolism of the central artery (August, Emrys Jones) may be present. Knapp has observed thrombosis of the central vein. The outcome in such cases is usually optic atrophy. Snell mentions hæmorrhages on the disc and in the vitreous. In 53 cases of erysipelatous orbital cellulitis Groenouw, 11 out of 47 died (Schwendt 11 out of 44), i.e. about 25 per cent. Of 24 unilateral cases of erysipelas 4 died (17 per cent.), and of 23 bilateral 7 (30 per cent.) (Schwendt 20 per cent. and 40 per cent. respectively). Iritis and iridocyclitis without corneal ulceration, and therefore probably metastatic, have been recorded by Hansen, Fortunati, Cornwell, Thier. Gillet de Grandmont found streptococci in the aqueous of a case with vitreous opacities.

Erysipelas may have a good effect upon ocular disease, e.g. trachoma (Baeck, Thier), phlyctenular keratitis (Thier), tuberculosis (Kuhnt), leprosy (Terson), simple and tubercular forms of uveitis (Walb, Nieden, Gayet, Zimmermann, Schmidt-Rimpler).

POLAND.—R. L. O. H. Rep., i, 1857. WEBER.—Med.-Chir. Trans., 1860. BIERMANN.—K. M. f. A., vii, 1869. PAGENSTECHER.—R. L. O. H. Rep., vii, 1870. HUTCHINSON.—R. L. O. H. Rep., vii, 1870; ix, 1879. WALB.—C. f. A., i, 1877. LUBINSKY.—K. M. f. A., xvi, 1878. LEBER.—A. f. O., xxvi, 3, 1880. NETTLESHIP.—Tr. Path. Soc., 1880; T. O. S., ii, 1882; R. L. O. H. Rep., xi, 1886. SCHWENDT.—Dissertation, Basel, 1882. VOSSIUS.—K. M. f. A., xxi, 1883. AUGUST.—K. M. f. A., xxii, 1884. EMRYS JONES.—Brit. Med. Jl., 1884. KNAPP.—A. f. A., xiv, 1885. NIEDEN.—C. f. A., ix, 1885. NEVE.—Brit. Med. Jl., 1886. RAMPOLDI.—Ann. di Ott., xv, 1886. DE SCHWEINITZ.—Philadelphia Hosp. Rep., 1890. GILLET DE GRANDMONT.—A. d'O., xii, 1892. ELSCHNIG, MITVALSKY.—K. M. f. A., xxxi, 1893. SCOUGAL, SNELL.—T. O. S., xiii, 1893. TERSON.—Ann. d'Oc., cxv, 1896. WAGENMANN.—B. d. o. G., 1896. ANDERSON CRITCHETT.—Ophth. Rev., xviii, 1899. WAGNER.—Ophth. Rec., 1899. BAECK, THIER, AXENFELD, RANSOHOFF.—K. M. f. A., xxxviii, 1900. KUHNT.—Z. f. A., iii, 1900. ZIMMERMANN.—Z. f. A., iv, 1900. ADDARIO.—A. di Ott., xii, 1904. CABANNÈS.—Rec. d'O., 1904. CARPENTER.—Ophth. Rec., 1904.

Anthrax (*malignant pustule*).—The anthrax pustule may occur on the lids; secondary affection of the lids or orbit from thrombosis of veins, etc., is rare. Elschnig has reported a rare case in which a woman was probably infected by her husband; almost invariably infection is derived directly from an animal. In 1077 cases the face was affected in 282, the lids in 10 (W. Koch), in 352 cases the upper lid in 2, the eyebrow in 3 (Thielmann). Anthrax pustule on the

conjunctiva is recorded by Knapp. Infection of the cornea has not been observed in man, and attempts to produce it in animals has led to discordant results (Frisch, Frank, Straus, and others).

HIMLY.—Die Krankheiten, etc., 1843. KNAPP.—A. f. A., v, 1876. W. KOCH.—Deutsche Chir., 1886. FRANK.—C. f. Bac., iv, 1888. STRAUS.—A. de Méd. exp., 1892. ELSCHNIG.—K. M. f. A., xxxi, 1893. SGROSSO.—Ann. di Ott., xxviii, 1898. RÖMER.—Vossius' Sammlung, iii, 1899. PRAUN AND PRÖSCHER.—C. f. A., xxiv, 1900. MOREAU.—Rev. gén. d'O., 1905.

Glanders.—Rare cases of primary infection of the lids with *Bacillus mallei* have been reported by Krajewski, Scheby-Buch, and Neisser. Differential diagnosis from syphilis or tubercle may be impossible without bacteriological examination (Neisser). Primary infection of the conjunctiva has been seen by Strzeminski and Kessler. Lacrymal fistula (Gourfein) and abscesses in the orbit (v. Graefe, Boyd) are reported. In animals ocular complications are not uncommon—111 times in 167 horses (Dupuy).

v. GRAEFE.—A. f. O., iii, 2, 1857. SCHEBY-BUCH.—Berliner klin. Woch., 1878. BOYD.—Trans. Path. Soc., xxxiii, 1883. NEISSER.—Berliner klin. Woch., 1892. TEDESCHI.—Ann. di Ott., xxi, 1892. GOURFEIN.—A. d'O., xxvii, 1897. STRZEMINSKI.—Z. f. A., v, 1901.

Actinomycosis.—The occurrence of actinomycosis of the lacrymal passages, frequently recorded, must be criticised in the light of recent researches (v. Vol. II, p. 759) : it is certain that other forms of streptothrix have been often mistaken for the *Streptothrix actinomyces*. Actinomycosis of the conjunctiva has been described by de Vincentiis, Demicheri, Fuchs. Secondary affection of the lids (Partsch), orbit (Ransom, Weeks), and changes in the eye following actinomycotic meningitis (Quervain, Bollinger) are open to less doubt. Inoculation of the anterior chamber in animals results in slight iritis (Ponfick, Dor).

See Vol. ii, p. 760. DE VINCENTIIS.—Lavori di Clin. oc. di Napoli, iii, 1893. DEMICHERI.—A. d'O., xix, 1899. FUCHS.—A. f. O., xlvi, 1, 1898. PARTSCH.—C. f. A., xvii, 1893. RANSOM.—Brit. Med. Jl., 1896. WEEKS.—New York Eye and Ear Infirmary Rep., 1897. QUERVAIN.—Deutsche Z. f. Chir., li, 1899. BOLLINGER.—Münchener med. Woch., 1887. PONFICK.—Virchow's Festschrift, Berlin, 1881. DOR.—Gaz. hebd., 1893.

Hydrophobia.—Primary infection through the lid (Nieden) and conjunctiva (Pentzold) is recorded. The virus is found after experimental inoculation in the lacrymal glands and vitreous, not in the aqueous (Högyes), though the animal can be infected by inoculation of the anterior chamber (Nocard and Roux).

NIEDEN.—C. f. A., iii, 1879. PENTZOLD.—Berliner klin. Woch., 1882. NOCARD AND ROUX.—Ann. de l'Institut Pasteur, 1888. HÖGYES.—In Nothnagel's Spec. Path., v, 2, 1897.

Trichinosis.—The *Trichina spiralis* is found in the eye muscles comparatively rarely (Kühn). Conjunctival œdema and even proptosis (Maurer) occur.

KÜHN.—Mitt. des Landwirtschaftlichen Instit. der Univ. Halle, Berlin, 1865. KITTEL.—Allg. Wiener med. Zeitung, 1871. MAURER.—Deutsches A. f. klin. Med., 1871.

Typhus fever.—Larionow collected cases of conjunctivitis, keratitis, iritis, retinitis, papillitis, night-blindness, etc., in typhus fever. In 253

cases of optic neuritis due to infectious diseases, three were caused by typhus (Uhthoft). Choroiditis (Hersing), metastatic ophthalmia (Mitvalsky), and bilateral dacryoadenitis (Lindner) are recorded.

CHISHOLM.—R. L. O. H. Rep., vi, 1867. TEALE.—Med. Times and Gaz., 1867. HERSING.—A. f. O., xviii, 2, 1872. LARIONOW.—K. M. f. A., xvi, 1878.

Typhoid fever.—Unusual dryness of the conjunctiva has often been noticed in typhoid fever (Berger) and keratomalacia has been attributed to this cause (Alt). Corneal ulcers are rare (Manz, Adler); they are probably due to ectogenous infection by pyogenic organisms since the cornea is little susceptible to typhoid bacilli (Gasparrini). In a case of typhoid with purulent keratitis, iridochoroiditis, retrobulbular cellulitis, and purulent dacryocystitis, Gasparrini found staphylococci and a bacterium which was either the typhoid bacillus or bacterium coli; the virulence of the latter is increased by the typhoid toxin, and it is responsible for many of the complications. Bilateral interstitial keratitis has been observed (Despagnet); herpes febrilis of the lids or cornea is rare. Iridochoroiditis (Hotz) and vitreous opacities (Larionow) have been seen. Metastatic infection may occur late, as in the case of Gillet de Grandmont, with iritis and hypopyon, the bacilli being found in the aqueous, and in the cases of panophthalmitis recorded by Millikin and Butten. Several cases of cataract have been reported (e.g. Campbell), usually in patients under 30. Typhoid fever is undoubtedly responsible for optic neuritis in some cases. Groenouw has collected 20 cases, and Uhthoff found 17 in 253 cases of optic neuritis due to infectious diseases. It occurs usually in the second or third week (e.g. Leber and Deutschmann), but may be later (two months, Munier). Both eyes are usually affected, but sometimes unequally (Seggel). There may be hæmorrhages near the macula (v. Petershausen). It is doubtful if primary atrophy occurs. The optic neuritis is generally secondary to cerebral mischief, as shown by the symptoms (Huguenin, Eisenlohr), but changes in the brain may be absent (Braine-Hartnell). Rosenberg obtained optic neuritis by injecting typhoid toxins into the subarachnoid space of rabbits. Central scotoma points to retrobulbar neuritis in some cases (König).

Amaurosis without disease of the nerve is very rare (Heddaeus—retinal ischæmia); some are probably due to loss of blood (Ebert, Williams). Snell recorded a case of embolism of the central artery. Retinal anæsthesia (Leber) and night-blindness (Larionow) are reported. Retinal (Bouchut, Gimurto), conjunctival, and orbital (Finlay) hæmorrhage occurs. Gasparrini found typhoid bacilli in an orbital abscess and Panas in a suppurating orbital angioma. Many cases of muscular paralyses are on record.

HEDDAEUS.—K. M. f. A., iii, 1865. EBERT.—K. M. f. A., vi, 1868. LEBER.—In G.-S., v, 1877. LEBER AND DEUTSCHMANN.—A. f. O., xxvii, 1, 1881. OGLESBY.—Brain, v, 1882. SNELL.—Ophth. Rev., i, 1882. WILLIAMS.—A. of O., xiii, 1884. HOTZ.—Am. Jl. of O., 1884. NETTLESHIP.—R. L. O. H. Rep., xi, 1886. DESPAGNET.—Ann. d'Oc., c, 1888. MANZ.—Münchener med. Woch., 1888. PANAS.—Progrès méd., 1891. ALT.—Am. Jl. of O., 1892, 1897. BERGER.—Les Maladies des Yeux, etc., Paris, 1892; Rev. gén. d'O., 1894. BRAINE-HARTNELL.—Brit. Med. Jl., 1897. BULL.—Med. Record, 1897. FINLAY.—A. f. A., xxxvii, 1898. FLEMMING.—Brit. Med. Jl., 1899. ANTONELLI.—Die ophth. Klinik, 1900; Ann. d'Oc., cxxv, 1901. CAMPBELL.—T. O. S., xx, 1900. RICHMOND.—Brit. Med. J.l, 1900.

DE SCHWEINITZ.—Philadelphia Med. Jl., 1900. OLIVER.—T. Am. O. S., 1902. SOURDILLE.— La Clinique ophth., 1903. CARPENTER.—Ophthalmoscope, 1904. PAUL.—K. M. f. A., xliv, 1906.

Relapsing fever.—Ocular disturbance was noted in this disease by Wallace (1826) and attributed to disease of the retina and choroid (Mackenzie). It is generally, however, cyclitis (Estlander, Logetschnikow), which may be acute—rarely simple iritis (Peltzer, Adamück)—or chronic. The frequency of uveitis varies from 2—12 per cent. Cyclitis has been produced experimentally in a monkey by inoculation of the *Spirochæta Obermeieri* (Ewetzky).

ESTLANDER.—A. f. O., xv, 2, 1869. LOGETSCHNIKOW.—A. f. O., xvi, 1, 1870. UHTHOFF.—Deutsche med. Woch., 1880. EWETZKY.—C. f. A., xxi, 1897. GROENOUW.— K. M. f. A., xxxviii, 1900; in G.-S., xi, 1, 1903.

Malaria.—Ocular complications occur in 10 per cent. of cases of malaria (Poncet). Apart from conjunctivitis corneal affections are common, and take the form of herpes febrilis with dendritic ulcers or interstitial keratitis (Poncet and Javal, Achenbach, Desvaux). Iritis, iridocyclitis, metastatic ophthalmia, retinitis pigmentosa, choroiditis, vitreous opacities, retrobulbar neuritis, cataract, glaucoma, etc., are described. Retinal hæmorrhage is relatively common and severe vitreous hæmorrhage may occur. Uhthoff found 17 cases of optic neuritis in 253 due to infectious diseases. It is doubtful if primary optic atrophy occurs. Anatomical investigations have been carried out by Guarnieri.

MACNAMARA.—Med. Times and Gaz., 1868; Brit. Med. Jl., 1890. PONCET.—Ann. d'Oc., lxxix, 1878. CARTER.—T. O. S., vi, 1886. KIPP.—T. Am. O. S., 1889. GUARNIERI.— A. per le Sc. med., xxi, 1897. YARR.—Brit. Med. Jl., 1898, 1899. *GROENOUW.—In G.-S., xi, 1, 1903 (Bibliography). SGROSSO.—A. di Ott., xiv, 1906.

Plague.—Conjunctivitis (Yamagiwa) and conjunctival pustules containing *Bacillus pestis* (Calmette and Salimbeni) occur in this disease. Corneal ulcers, interstitial keratitis, iritis, iridocyclitis (Rees), panophthalmitis, retinal hæmorrhage (Maynard), etc., are recorded. Römer produced plague in an animal by instillation of cultures of the bacillus into the conjunctival sac.

YAMAGIWA.—Virchow's Archiv, cxl, 1897. CALMETTE AND SALIMBENI.—Ann. de l'Institut Pasteur, 1899. RÖMER.—Z. f. Hygiene, xxxii, 1899. MÜLLER.—In Nothnagel, v, 1, Wien, 1900 (Bibliography). MAYNARD.—Brit. Med. Jl., 1901. REES.—Lancet, 1905.

Asiatic cholera.—Early cyanosis of the lids and extreme dryness of the conjunctiva from diminished secretion of tears are characteristic of cholera (v. Graefe, Joseph); conjunctival hæmorrhage is common. In severe cases necrosis of the lower part of the cornea, due to keratomalacia, occurs. Iritis and choroidal hæmorrhage have been reported (Williams). Vitreous opacities and cataract are to be attributed to disease of the uveal tract. Metastatic ophthalmia has not been observed with certainty (Axenfeld). The retinal arteries are extraordinarily narrowed and dark in colour, whilst an arterial pulse or cessation of circulation is easily produced by slight external pressure upon the globe; a broken blood-column has been observed (v. Graefe). The veins are normal in calibre, but extremely dark in colour.

Joseph.—Günsburg's Zeitschrift, vii, 1856. v. Graefe.—A. f. O., xii, 2, 1866. Williams.—St. Louis Med. and Surg. Jl., 1885. Delens.—A. d'O., vi, 1886. Webster Fox.—Med. Bull., 1893.

Tetanus.—Cramp of the orbicularis (Samelsohn) and of extrinsic ocular muscles (Fromaget) occurs in tetanus. Paralysis of the orbicularis is always part of general paralysis of the facial nerve.   Paralysis of the third nerve, with or without the pupillary branch and with or without implication of other nerves, has been observed several times (Roberts, Marx, Rockliffe).   Optic neuritis (Hughlings Jackson) is rare.   The eye is seldom the primary site of entry of the bacillus, 9 cases only being on record, but the primary lesion is not infrequently near the eye.   Orbital wounds giving rise to tetanus have been collected by Fromaget.

Hughlings Jackson.—Med. Times and Gaz., 1878. Samelsohn.—C. f. A., iii, 1879. Chisholm.—A. of O., ix, 1880. Hotz.—Chicago Med. Rev., 1882. Robinson and Hooker.—Lancet, 1883. Bernhardt.— Z. f. klin. Med., 1884. Rockliffe.—Brit. Med. Jl., 1890. Roberts and Williamson.—Lancet, 1891. Rust.—Jl. of Ophth., Otol., and Laryn., 1892. Fromaget.—A. d'O., xiv, 1894 (Bibliography). Kepiner.—Ann. of O., 1895. Mayer.—Wiener med. Woch., 1901. Genth.—Z. f. A., ix, 1903.

Influenza.—Ocular complications of influenza occur in 7 per cent. of the cases (Leyden and Guttmann).   Groenouw has collected 186 cases in which the distribution of the ocular affection was as follows:— Eyelids 4·3 per cent., conjunctiva 29·6 per cent., cornea 31·2 per cent., uveal tract 8·1 per cent., retina 3·2 per cent., optic nerve 4·8 per cent., vitreous 1·6 per cent., lens 2·2 per cent., eye muscles 8·6 per cent., glaucoma 2·7 per cent.   Lid affections include œdema, blepharitis, abscesses—hordeola especially frequently—herpes, and whitening of the lashes (Bock).   Conjunctivitis is very common and may be pseudomembranous (Pflüger, zur Nedden, Coppez, Valude) ; besides the influenza bacillus streptococci have been found (Valude, zur Nedden).   Phlyctenular conjunctivitis and conjunctival haemorrhage are reported. Inflammation of the nasal mucous membrane is often associated with dacryocystitis (Greeff, Wicherkiewicz, Pergens), but the latter may be primary (Badal and Fage).   Dacryoadenitis has been observed several times (Lindner 1 case, Mohr 3 cases, Pignatari 16 cases).

Herpes corneæ is relatively frequent (Adler, Eversbusch, Fuchs, Greeff, Haab, Hirschberger, Pflüger, Sattler, Valude, Lindner, and others) ; it commences on the third to seventh day and is usually unilateral.   Similarly Fuchs's superficial punctate keratitis often follows influenza (Pflüger, Rosenzweig, Ehrlich) ; it usually commences at the end of the first week and has been accompanied by the unusual complication iritis.   Interstitial keratitis has several times been attributed to influenza (Adler, Wagenmann, Achenbach, Hilbert, Pflüger).   Hypopyon ulcer is not uncommon (Coppez, Adler, Badal, Denti, Eversbusch, Königstein, Rampoldi, Simi, Lindner, Mohr, McKeown).   Rare complications are neuroparalytic keratitis (Novelli, Lavagna) and keratomalacia (Ringier).

Iritis and iridocyclitis are common, choroiditis infrequent.   Cases of uveitis are recorded by Adler, Badal and Fage, Braunstein, Delacroix, Denti, Guttmann, Pflüger, Ehrlich, Truc, Natanson, and

others. Metastatic ophthalmia has been reported in 20 cases (Berlin, Rampoldi, Bull, Pflüger; with pneumonia—Blessig, Braunstein, Hosch, Natanson, Hirschmann, Desbrières, Eversbusch; with endo-carditis, purulent meningitis, and nephritis—Alfieri). Bacteriological examination has proved the presence of *Staphylococcus aureus* (Eversbusch), *Staphylococcus citreus* and another coccus (Lavagna), pure culture of influenza bacillus (Tanja): most cases are to be attributed to pyogenic organisms, not to the influenza bacillus. In one case the aqueous was sterile (Laqueur).

Vitreous opacities, possibly due to hæmorrhage, are frequently recorded, and lenticular opacities have been attributed to influenza without sufficient testimony. Retinal hæmorrhages (Gillet de Grandmont, Rampoldi, Galezowski, Ehrlich), embolism of the central artery (Coppez, Hillemanns, Hosch, Leyden, Guttmann), retinal ischæmia (Alt), detachment of the retina (Ringier), glaucoma (Rampoldi, Adler, Badal and Fage, and others), have been recorded.

Uhthoff in 253 cases of optic neuritis due to infectious disease found 72 attributed to influenza. Groenouw has collected 67 cases (Gazis, Landsberg, Lindner, Rampoldi, Stöwer, Novelli, Antonelli, Snell, Galezowski, Despagnet, Parinaud, de Schweinitz, Natanson, Pflüger, Schmidt-Rimpler, Remak, Hillemanns, Denti, Braunstein, Cross, Hartridge, Hirschmann, Wingenroth, Bergmeister, Hansen, and others). The complication is usually bilateral, and occurs at any age (6 years Natanson, 67 Schmidt-Rimpler). Papillitis may be associated with retinal hæmorrhages (Denti, Braunstein), star figure at the macula (Cross, Hartridge, Hirschmann, Wingenroth), disease of the vessel walls (Bergmeister), embolism of the central artery (Hansen). Primary optic atrophy has been reported (v. Schröder and others). Retrobulbar neuritis without ophthalmoscopic signs is infrequent. Optic neuritis is probably due in most cases to the direct action of the influenza toxins, but it may follow meningitis (Guttmann, Remak), albuminuria (McHardy), orbital periostitis, etc., and it may be associated with multiple neuritis (Braunstein, Gowers).

Simple or purulent Tenonitis has been recorded in 11 cases (Fuchs, Mohr, Greeff, Pflüger, Hodges, Schwarz, Schapringer, and others). It is usually unilateral (Greeff), and only rarely purulent (Fuchs). Orbital abscess has been recorded in 9 cases (Pergens, Lefrancois, Mohr, Borthen, Socor, Zimmermann, Valude, Siegrist). It is generally unilateral, and most of the patients have been young. Five cases have been examined bacteriologically, streptococci, staphylococci, and pneumococci being usually found; in Siegrist's case the influenza bacillus was present alone.

Visual defects—hemianopia (Albrand, Gifford, Gradly), amblyopia etc.—and paralysis of extrinsic muscles have been frequently found.

BOCK.—C. f. A., xiv, 1890; K. M. f. A., xxviii, 1890. v. SCHRÖDER.—St. Petersburger med. Woch., 1889, 1892. ADLER.—Wiener med. Woch., 1890. ALT.—Jl. of O., 1890. BADAL AND FAGE.—A. d'O., x, 1890. COPPEZ.—Rev. gén. d'O., 1890. DENTI.—Ann di Ott., xix, 1890. EVERSBUSCH.—Münchener med. Woch., 1890. FUCHS.—Wiener klin. Woch., 1890. GALEZOWSKI, GAZIS, GILLET DE GRANDMONT.—Rec. d'O., 1890. GREEFF, LAQUEUR.—Berliner med. Woch., 1890. GUTTMANN, HOLZ, PFLÜGER.—Berliner klin. Woch., 1890. HAAB, HOSCH.—Korrespondenzbl. f. Schweizer Aerzte, 1890. LANDSBERG,

REMAK, ROSENZWEIG.—C. f. A., 1890. MARCHISIO, RAMPOLDI.—Ann. di Ott., xix, 1890. NATANSON.—St. Petersburger med. Woch., 1890; Die ophth. Klinik, 1900; K. M. f. A., xxxix, 1901. SCHIRMER, STÖWER.—K. M. f. A., xxviii, 1890. UHTHOFF.—Deutsche med. Woch., 1890. BORTHEN.—K. M. f. A., xxix, 1891. LINDNER.—Wiener med. Woch., 1891. NOVELLI.—Boll. di Ott., 1891. WEEKS.—New York Med. Jl., 1891. ANTONELLI.—Ann. di Ott., xxi, 1892. GRADLY.—Ophth. Rec., 1892. HARTRIDGE, HODGES.—Ophth. Rev., xi, 1892. CROSS.—T. O. S., xiii, 1893. LEYDEN AND GUTTMANN.—Die Influenzaepidemie 1889—1890, Wiesbaden, 1892. SNELL.—T. O. S., xii, 1892. GOWERS.—Lancet, 1893. PIGNATARI.—Rev. gén. d'O., 1894. PERGENS, GALEZOWSKI.—Ann. d'Oc., cxiii, cxiv, 1895. PFLÜGER, WAGENMANN.—B. d. ö. G., 1896. HILBERT.—Die ophth. Klinik, 1898. VALUDE.—Ann. d'Oc., cxix, 1898. FLEMMING.—Brit. Med. Jl., 1899. LEFRANÇOIS.—A. d'O., xix, 1899. WINGENROTH.—K. M. f. A., xxxvii, 1899. ZUR NEDDEN.—K. M. f. A., xxxviii, 1900; xli, 1903. BULL.—T. Am. O. S., 1901. DE SCHWEINITZ.—Ophth. Rec., 1901. MCKEOWN.—Brit. Med. Jl., 1902. *GROENOUW.—In G.-S., xi, 1, 1903 (Bibliography).

POISONS.

The chief ocular complication produced by various poisons is amblyopia, commonly known as toxic amblyopia, to which special attention will be directed here.

DE SCHWEINITZ. The Toxic Amblyopias, Philadelphia, 1896. *UHTHOFF.—In G.-S., xi, 2, 1901. *LEWIN AND GUILLERY.—Die Wirkungen von Arzneimitteln u. Giften auf das Auge, Berlin, 1905.

Alcohol and tobacco.—Amblyopia from alcohol occurs almost exclusively in men, but women are not exempt in the rarer cases in which they come under the influence of the poison. Uhthoff found 10 per cent. women in East Germany. Beer and wines seldom induce the condition ; it is generally due to spirits, especially those containing impurities such as fusel oil.

Acute alcohol amblyopia has been most frequently described in poisoning with methyl alcohol (Mengin, Viger, Moulton, Gifford, Patillo, Kuhnt, McKoy and Michael, Casey Wood). Severe amblyopia or complete amaurosis sets in rapidly; central scotoma is seen as a rule only in the stage of retrogression in those cases in which optic atrophy does not ensue. Occasionally there is optic neuritis in the early stages (McKoy and Michael, Hotz). Smaller doses may cause chronic amblyopia, but seldom the typical picture associated especially with ethyl alcohol and tobacco. Men are more susceptible than animals, but experiments on the latter (Ward Holden, Birch-Hirschfeld) show characteristic changes in the retinal ganglion cells and optic nerve-fibres. Birch-Hirschfeld, from experiments on rabbits and fowls, concludes that the primary lesion is in the ganglion cells, which show marked degenerative changes, and that the degeneration of the nerve-fibres is secondary, evidence of inflammatory and vascular changes being absent.

BOERHAVE.—Abhandlung von Augenkrankheiten, Nürnberg, 1771. VIGER.—L'anneé méd., 1877. MENGIN.—Rec. d'O., 1879. THOMPSON.—Med. and Surg. Rep., 1897. MCKOY AND MICHAEL.—Med. Rec., 1898. GIFFORD.—Ophth. Rec., 1899, 1906. WARD HOLDEN.—A. of O., xxviii, 1899. KUHNT.—Z. f. A., i, 1899. MOULTON, PATILLO, RAUB, WOODS, COLBURN.—Ophth. Rec., 1899. WOOD, HOTZ.—Chicago Ophth. and Otol. Soc., 1899. STIEREN.—Am. Med. Assoc., 1900. *BIRCH-HIRSCHFELD.—A. f. O., lii, 1901; liv, 1902. *UHTHOFF.—In G.-S., xi, 2, 1901 (Bibliography). RING.—T. Am. O. S., 1902. BULLER, WOOD.—Am. Med. Assoc., 1904. BRUNER.—Ophth. Rec., 1904. STIRLING.—Ophth. Rev., xxiv, 1905. DE SCHWEINITZ.—T. Am. O. S., 1907. STRÖHMBERG.—St. Petersb. med. Woch., 1904.

Chronic alcohol amblyopia is often associated with tobacco ambly-opia, and it is difficult to distinguish the relative importance of the two factors.   There is no doubt that either poison can produce the typical clinical picture alone; thus Uhthoff in 327 cases of toxic amblyopia found 41 of pure tobacco amblyopia, the remaining 286 being due principally to alcohol or to the combined use of alcohol and tobacco.   The curve of age-incidence rises to a maximum at 45 years (88 per cent.).   The well-known symptoms nearly resemble those of retrobulbar neuritis. It is doubtful if complete optic atrophy has ever been observed as the result of uncomplicated alcohol or tobacco amblyopia ; Uhthoff has never found such a case.   On the other hand, cases complicated by hereditary optic neuritis leading to optic atrophy are on record, and tabes and other causes of atrophy must be eliminated.   Optic atrophy ascribed to alcohol and tobacco is reported by Hutchinson, Wordworth, Deneffe, Sichel, Berry, and others.   The absence of a relative central scotoma is extremely rare (*e. g.* Vossius) : a small absolute central scotoma is occasionally observed, and concentric contraction of the field is also rare.   The condition is probably always bilateral.   The temporal side of the disc is generally paler than normal in cases which have persisted for some weeks.   Uhthoff in 11 cases examined anato-mically found that the pallor bore a definite relationship to the amount of degeneration in the nerve.   In the early stages there may be slight hyperæmia and obscuration of the edges of the disc (8 per cent. of cases, Uhthoff) ; it is noteworthy that slight optic neuritis is not very uncommon in alcoholism with polyneuritis (Rennert, Thomsen, Lilien-feld, Sachs, Strümpell, Gudden, Miles Standish, and others), polio-encephalitis, etc.   Retinal changes which have been observed—white plaques, hæmorrhages, vascular changes, etc.—are probably due to some intercurrent cause (Lawford, Jaeger, Ord, Berry, and others).

A large number of cases have now been submitted to anatomical investigation (Erismann, Leber, Samelsohn, Nettleship and Edmunds, Vossius, Bunge, Sachs, Widmark, Schmidt-Rimpler, Magnan, Stoelt-zing, de Schweinitz, Edmunds and Lawford, Boedecker, Sourdille, Siegrist, Nuel, Uhthoff, Birch-Hirschfeld).   In all the essential change has been degeneration of the papillo-macular bundle.   In passing from before backwards along the nerve the bundle is first wedge-shaped and temporal, then crescentic and situated down and out, then round and axial, and finally before entering the chiasma transversely oval and axial.   The degeneration can be traced along the optic tracts to the so-called primary optic centres.   The degeneration can by no means always be traced throughout the whole extent of this course.   It often disappears in the posterior segments ; it may be most pronounced at the optic foramen (Samelsohn and others), thus giving rise to the view that the primary lesion is situated here ; in other cases it is most marked immediately behind the globe.   Examination of the retina has shown changes in the ganglion cells, not limited to the macular region, and atrophy of the nerve-fibres, as well as less obvious changes in the nuclear layers (Nuel, de Schweinitz, Widmark, Siegrist, Birch-Hirsch-feld, and others).   Uhthoff and others consider that these changes could well be accounted for as secondary to a retrobulbar neuritis, and

this is undoubtedly true. They could also, I think, even more reason-ably be attributed to primary poisoning of the ganglion cells, resulting in secondary degeneration of the nerve-fibres, and Birch-Hirschfeld has brought forward very potent evidence in favour of this view. It is supported by the analogy of the results of experimental poisoning with methyl alcohol and other drugs, *e. g.* filix mas, and an exhaustive examination of a case of tobacco amblyopia by the Nissl method shows that there is no evidence of retrobulbar inflammation, but that the changes in the nerve are best accounted for on the theory of secondary degeneration (Birch-Hirschfeld).

Primary changes in the blood-vessels have been described (Sourdille, de Schweinitz, Sachs, and others). Sachs lays stress upon changes in the distribution of the posterior central vein, Samelsohn and Vossius on those in the neighbourhood of the optic foramen. Much discussion centres around the changes in the neuroglia, Nuel regarding this as primary, whilst Birch-Hirschfeld considers that the proliferation is minimal and purely secondary to the degeneration of the nerve-fibres.

Much experimental investigation on animals has been directed towards elucidating the pathology of the disease (Ludger, Lallemand, Perin and Duroy, Pupier, Ruge, Cremiansky, Magnan, Holden, de Schweinitz, Birch-Hirschfeld, and others). It must be admitted that the results have been somewhat disappointing, and this may be largely attributed to the difficulty of reproducing the effects of chronic poison-ing. The most successful experiments have been those on methyl alcohol, the acute condition being reproduced with comparative ease.

Among the rarer complications of alcoholic amblyopia are para-lysis of the extrinsic muscles, *e. g.* abducens (Leprince), and ophthalmo-plegia externa, generally attributed to hæmorrhagic polio-encephalitis superior (Thomsen, Boedecker, Oppenheim, Suckling, Wilbrand and Saenger, and others). Nystagmus and disorders of the pupillary reactions have been described.

Tobacco amblyopia has already been considered in relation with alcoholism. Tobacco amblyopia was first described by Beer (1817), but the observation appears to have been overlooked. It was resusci-tated by Mackenzie (1854), and amply confirmed later by Sichel, Wordsworth, Hutchinson, Foerster, Hirschberg, Leber, Nettleship, and others. There is no doubt that it may occur in the absence of alcoholism. It is certain, too, that women addicted to tobacco may be attacked (Priestley Smith, de Schweinitz, Chisholm, Lyder Borthen, Berry, Hill, Griffith, Nettleship, and others). It is noteworthy that the patients may have been long excessive smokers without evil effect, but are attacked during some depression of the general health—defective nutrition from loss of employment, worry, etc. The patients are usually between 30 and 50 years of age. In younger patients there has in several instances been a predisposition to hereditary optic neuritis (Habershon, Browne, Hormuth).

The clinical features are indistinguishable from those of combined alcohol and tobacco amblyopia. Cases in which optic atrophy has supervened (Sichel, Wordsworth, Hutchinson, Drysdale, Panas,

Lawford, Frost, and others) are probably not due to simple misuse of the drugs. In England the sufferers usually smoke "shag," but tobacco mixtures may also cause amblyopia, less frequently cigars, and very rarely cigarettes. The condition may be caused by, or kept up by, tobacco chewing. Statistics give a percentage of 0·04—0·13 of all eye patients (Uhthoff 0·07 per cent.) : the condition appears to be commoner in some countries, *e. g.* England, than in others.

Amblyopia is produced in horses by feeding upon certain plants, notably *Nicotiana suaveolans* in Australia (Husemann, Barrett, de Schweinitz).

Rarer complications are paralyses of extrinsic muscles (Dalichow, Fontan, Jan), Argyll-Robertson pupil, miosis with retained light reaction (Tamassia, Gysi, Rava, and others), etc.

It has been too readily concluded that tobacco amblyopia is caused by nicotin. The nicotin content of various tobaccos varies from 2—8 per cent. Nicotin is an extremely potent poison but it is only slightly volatile, whilst other equally strong poisons, more highly volatile, are found in tobacco smoke in small quantities, such as pyridin (C_5H_5N), and its derivatives picolin ($C_5H_4(CH_3)N$), lutidin ($C_5H_3(CH_3)_2N$), collidin ($C_5H_2(CH_3)_3N$), etc. These drugs might well repay experimental physiological investigation. The effect of nicotin upon nerve-cells has received much attention since Langley discovered that it paralysed them after a preliminary stage of excitation. The effect of administering the drug to animals has been investigated histologically for the cells of the central nervous system by Vas and Pándi. The paralysing effect is obtained whether the drug is injected into the circulation or applied to the cells locally. My experiments on the superior cervical ganglion show that local application of nicotin in moderate concentration produces no histological change which is demonstrable by the Nissl method, the most delicate at our disposal. This supports the conclusion arrived at by Langley that the drug acts upon the branchings (synapse) of the preganglionic fibres around the cells rather than upon the cell itself. Warrington has shown that cutting off the afferent impulses to a nerve-cell is an even more potent cause of degeneration of the Nissl granules than cutting its own axis cylinder process. If these conclusions are applied, with all reservation, to toxic amblyopia, it would appear that the poison acts upon the synapses, either of the cone fibres or of the cone bipolars, or both.

The explanation of the special vulnerability of the macular region is more difficult. From Birch-Hirschfeld's investigations it is seen that in experimental toxic amblyopia degenerative changes occur in ganglion cells in all parts of the retina, but these results must not be pressed too far as an analogy to the very chronic clinical condition. The special differentiation of the macular region finds its anatomical expression in an "individualisation" (Ramon y Cajal) of the nerve elements, whereby each ganglion cell is related to a single cone bipolar and cone, not to several as in other parts of the retina. This differentiation is a sign of greater complexity in the terms of Herbert Spencer, and therewith of greater vulnerability. Without, however, having recourse to a teleological explanation the failure of the papillo-macular

system may be due to vascular causes. These undoubtedly play an enormous part in the pathology of quinine amblyopia (Ward Holden), but the analogy is very imperfect, both on clinical and ophthalmoscopic grounds. My experiments on the ocular circulation have shown that large doses of nicotin, applied rapidly, cause an enormous rise of blood-pressure due to stimulation of all the sympathetic ganglia in the body, and this leads to a passive dilatation of the intra-ocular vessels. It does not follow that this passive dilatation will occur with minute doses spread over a long period. It is further probable that the sympathetic ganglia are much more resistant to toxic effects than such highly differentiated cells as those of the retina, and above all, those of the macula. It is therefore probable that the retinal cells will give evidence of the paralytic and degenerative change earlier than the sympathetic ganglia. In fact, the retinal cells, and especially the macular cells, will be in the paralytic stage whilst the sympathetic cells are still in the stage of excitation. It is certain that an active local constriction can be obtained with small doses of drugs, such as adrenalin and nicotin ; and it is most likely that this will be the result in chronic poisoning, thus affording an additional reason for the vulnerability of the macula. Possibly, too, the poor vascular supply of the macula from the retinal circulation is of importance, though this will be abolished if both the retinal and ciliary systems suffer equally, for the choroidal blood supply is especially good in the macular region.

BEER.—Lehre von den Augenkrankheiten, ii, 1817. MACKENZIE.—Treatise, 4th Ed., 1852. HART, HUTCHINSON, WORDSWORTH.—Lancet, 1863. SICHEL.—Ann. d'Oc., liii, 1865, ERISMANN.—Dissertation, Zürich, 1867. HUTCHINSON.—Med.-Chir. Trans., 1867; R. L. O. H. Rep., vii, 1871; viii, 1876. NUEL.—Ann. d'Oc., lxxx, 1878; Internat. Congress, Paris, 1900. BERRY.—R. L. O. H. Rep., x, 1880; Ophth. Rev., iii, 1884. SWANZY.—Med. Rec., 1881. BORTHEN.—Rec. d'O., 1882. LAWFORD, MORTON.—Lancet, 1882. NETTLESHIP AND EDMUNDS.—T. O. S., ii, 1882. SAMELSOHN.—A. f. O., xxviii, 1, 1882. VOSSIUS.—A. f. O., xxviii, 3, 1882; K. M. f. A., xxi, 1883. PRIESTLEY SMITH, SUCKLING.—Brit Med. Jl., 1883. SHEARS.—Brit. Med. Jl., 1884. AYRES.—Am. Jl. of O., 1885. BULL.—New York Med. Jl., 1886. HILL GRIFFITH, HARTRIDGE.—Brit. Med. Jl., 1886. UHTHOFF.—A. f. O., xxxiii, 1, 4, 1886. HUTCHINSON, JR.—R. L. O. H. Rep., xi, 1886. NETTLESHIP.—R. L. O. H. Rep., xi, 1886. DISCUSSION ON TOXIC AMBLYOPIA.—T. O. S., vii, 1887. *DE SCHWEINITZ.—Med. News, 1886; The Toxic Amblyopias, Philadelphia, 1896; T. Am. O. S., 1897; Ophth. Rec., 1897, 1900; T. Am. O. S., 1907. BROWNE, HABERSHON.—T. O. S., viii, 1888. CHISHOLM.—Am. Jl. of O., 1887. SACHS.—A. f. A., xviii, 1887; xxvii, 1893. THOMSEN.—A. f. Psych., xix, 1887; xxi, 1889. *UHTHOFF.—A. f. O., xxxii, 4; xxxiii, 1, 1887; in G.-S., xi, 2, 1901 (Bibliography). DOYNE.—R. L. O. H. Rep., xii, 1888. SUCKLING.—Brit. Med. Jl., 1888. EDMUNDS AND LAWFORD.—T. O. S., ix, 1889. LAWFORD.—T. O. S., x, 1890. PRIESTLEY SMITH.—Brit. Med. Jl., 1890. ABNEY.—Proc. Royal Soc., xix, 1891. GROENOUW.—A. f. O., xxxviii, 1, 1892; xl, 2, 1894. PÁNDI.—Ungar A. f. Med., iii, 1893. WOOD.—Ann. of Ophth. and Otol., i, 1892. VAS.—A. f. exp. Path., 1894. BOEDECKER.—A. f. Psych. xxvii, 1895. SPICER.—T. O. S., xv, 1895. A. H. THOMPSON.—R. L. O. H. Rep., xiv, 1896. WIDMARK.—Neurol. Centralblatt, 1897. BARRETT.—Ophth. Rec., 1897. BRUNTON.—Brit. Med. Jl., 1900. SOURDILLE.—Ophth. Klinik, 1900. SIEGRIST.—A. f. A., xli, 1900. BIRCH-HIRSCHFELD.—A. f. O., l, 1900: lii, 1901; liii, 1901; liv, 1902; Ophthalmoscope, 1904. *PARSONS.—Brit. Med. Jl., 1901; Ophth. Rev., xx, 1901; The Ocular Circulation, London, 1903. ZENTMAYER.—Ann. of O., 1901. SCHIECK.—A. f. O., liv, 1902; lvi, 1903. STIRLING.—Ophth. Rev., xxiv, 1905. MARIE AND LÉRI.—Rev. neurol, 1905. BÄR.—A. f. A., liv, 1906. DALÉN.—Mitt. a. d. Augenklinik zu Stockholm, 1906. TOJODA, SCHOLTZ.—K. M. f. A., xlv, 1907.

Carbon disulphide.—This substance produces amblyopia resembling that associated with alcohol and tobacco. Carbon disulphide is used extensively as a solvent for gutta-percha. Central vision is usually

most affected, the field showing a central scotoma for red and green, rarely peripheral contraction as in the cases reported by Little, Maas, Kalischer, Lavigerie, Ross. The prognosis is fairly good ; blindness is very rare, but considerable amblyopia with partial optic atrophy may persist. Ophthalmoscopically there is slight optic neuritis (Delpech, Nuel and Leplat, Gowers, Frost) or temporal pallor of the disc (Frost, Kalischer, Becker). General pallor of the disc is rare (Lavigerie). Absence of change in the fundus has led to the diagnosis of retro-bulbar neuritis (Edge, Gallemaerts). Absolute central scotoma is commoner than in alcohol and tobacco amblyopia. Hirschberg describes slight changes at the macula. No anatomical examination in man has been recorded. Experimental observations show changes in the cells of the central nervous system (Koester, Birch-Hirschfeld), but none in the retina (Birch-Hirschfeld).

Delpech.—L'Union, 1856 ; Nouvelles Recherches, etc., 1863. Galezowski.—Rec. d'O., 1877. Bruce.—Edin. Med. Jl., 1884. Nettleship.—Med. Times and Gaz., 1884. Frost, Fuchs.—T. O. S., v, 1885. Marcus Gunn.—T. O. S., vi, 1886. Hirschberg.—C. f. A., x, 1886 ; xiii, 1889. Lavigerie.—Rec. d'O., 1887. Ross.—Lancet, 1887. Becker.—C. f. A., xiii, 1889. Edge.—Lancet, 1889. Nuel and Leplat.—Ann. d'Oc., ci, 1889. Gallemaerts.—Ann. d'Oc., civ, 1890 ; Polyclinique, 1898. Kalischer.—Allg. med. Centralzeitung, 1896. de Schweinitz.—The Toxic Amblyopias, Philadelphia, 1896. Laudenheimer.—Neurol. Centralblatt, 1898. Koester.—A. f. Psych., xxxii, 1899. Remak.—Neuritis u. Poly-neuritis, Wein, 1900. Birch-Hirschfeld.—A. f. O., l, 1900. *Uhthoff.—In G.-S., xi, 2, 1901.

Iodoform.—Iodoform amblyopia usually resembles alcohol and tobacco amblyopia in the presence of a central scotoma, free peripheral field, temporal pallor of the disc, etc. (Hirschberg, Hutchinson, Priestley Smith, Terson, Anderson Critchett). In some cases there is transitory amaurosis (Valude), unilateral narrowing of the field, etc.

Hirschberg.—C. f. A., vi, 1882. Hutchinson.—New York Med. Jl., 1886. Priestley Smith.—Ophth. Rev., xii, 1893. Valude.—Ann. d'Oc., cix, 1893. Russell.—Lancet, 1893. Terson.—Ann. d'Oc., cxviii, 1897 ; A. d'O., xvii, 1897. Anderson Critchett.—T. O. S., xviii, 1898. Brose.—A. of O., xxix, 1900. de Vries.—Neederl. Tijdsch. v. Geneesk., 1901. *Mohr.—A. f. A., xlv, 1902 (Bibliography). Palermo.—Ann. di Ott., xxxiv, 1905.

Quinine.—Quinine amblyopia shows important differences from that produced by alcohol and tobacco. There is either complete amaurosis or peripheral contraction of the field without central scotoma. The condition is acute and is induced by very variable doses of the drug, idiosyncrasy playing an essential part. Gowers states as the minimal dose 5 grms. within 30 hours, and 78 grms. within 3 days as the maximal. The toxic dose in dogs is given by Holden as 0·07 grms. per kilo. body weight, by de Schweinitz as 1—4 grms. per pound. Transitory amblyopia associated with deafness, palpitation, etc., is not uncommon after quinine ; severe amblyopia must be regarded as rare. The disease is always bilateral, the earliest recorded case by v. Graefe being probably due to the malaria, not the quinine. Transitory amaurosis has been frequently recorded, always accompanied by deafness. Central vision is first restored, but permanent con-traction of the field in greater or less degree is the rule. Persistent blindness is rare. Central scotomata have been reported by Jodko and

Bietti; partial eccentric scotomata by Mellinger and de Wecker. The colour field shows nothing characteristic, colour perception being depressed with light and form perception. Panas records night-blindness. Ophthalmoscopically the striking features are extreme contraction of the retinal vessels and pallor of the disc. In the earliest stage the picture of obstruction of the central artery of the retina may be perfectly reproduced.

There can be no doubt that the essential factor in the disorder is the retinal ischæmia. Whether the drug also depresses the activity of the retinal cells is less certain. In favour of this view is the observation of de Bono that quinine causes diminution in the movements of the pigment cells under the influence of light in frogs. Whether the constriction of the retinal vessels is active (Adamük, Monteverdi) or passive has been disputed. My own experiments confirm those of many other investigators that it is passive. Intra-venous injection of quinine causes extreme dilatation of the vessels of the splanchnic area associated with passive constriction of those of the eye and other peripheral areas. Since quinine amblyopia is an acute condition in its onset these experimental results may be applied to the clinical condition with confidence.

Degeneration of the inner layers of the retina and especially of the ganglion cells has been found by Holden, Druault, and Altland experimentally. The chromatolysis of the cells is followed by degeneration of the nerve-fibres, and there can be no doubt that extensive optic atrophy occurs in all but the transient cases observed clinically. It is impossible to decide at present whether this results simply from defective blood supply or from this cause associated with direct poisoning of the cells.

v. Graefe.—A. f. O., iii, 2, 1857. Adamük.—K. M. f. A., vi, 1868. Lewitzky.—Virchow's Archiv, xlvii, 1869. Kerner.—Pflüger's Archiv, 1870. Lightfoot.—Brit. Med. Jl., 1870. Roosa.—Am. Jl. of Med. Sc., 1874; New York Med. Rec., 1878; T. Am. O. S., 1887. Buller.—T. Am. O. S., 1881. Grüning, Knapp, Michel.—A. f. A., xi, 1882. Webster.—Arch. of Med., 1883. Nettleship, Browne.—T. O. S., vii, 1887. Mellinger.—K. M. f. A., xxv, 1887. Schulz.—Virchow's Archiv, cix, 1887. Ulrich.—A. f. O., xxxiii, 2, 1887. Peschel.—Ann. di Ott., xvi, 1887. Barabaschew.—A. f. A., xxiii, 1891. de Schweinitz.—T. Am. O. S., 1891; Ophth. Rev., x, 1891; The Toxic Amblyopias, Philadelphia, 1896; Ophth. Rec., 1898; 1899; T. Am. O. S., 1907. de Bono.—Arch. di Ott., ii, 1894; vi, 1899. Claiborne.—Med. Rec., 1894. Roberts.—Lancet, 1895. Demicheri.—Ann. d'Oc., cxv, 1896. Ellis, Ayres, Dickson.—Am. Jl. of O., 1897. Colhoun, Harlan.—Ophth. Rec., 1897. Santos Fernandez.—Jl. of Eye, Ear, and Throat Dis., 1897. Holden.—T. Am. O. S., 1898; A. of O., xxvii, 1898. Druault.—Internat. Congress of Med., 1900; A. d'O., xxii, 1902. Birch-Hirschfeld.—A. f. O., l, 1900. Schwalbe.—A. f. A., xlii, 1900. Nohl.—B. z. A., xlviii, 1901. Schoute.—Z. f. A., ix, 1903. Stölting.—A. f. O., lv, 1903. Parsons.—The Ocular Circulation, London, 1903. Altland..—K. M. f. A., xlii, 1904. Miller.—Brit. Med. Jl., 1905. Vermes.—Z. f. A., xiv, 1905. Parker.—A. of O., xxxv, 1906. Seeligsohn.—C. f. A., xxx, 1906. Culbertson.—Am. Jl. of O., 1906.

Salicylic acid.—Salicylic acid and salicylates produce disorders of vision and hearing similar to those of quinine (Leber, Buss, Ries, Knapp, de Schweinitz), though much less often and of less intensity. The characteristic ophthalmoscopic changes (Knapp in men, de Schweinitz in dogs) are seldom seen. Such cases as that of Gatti in which 8 grms. of salicylate taken in 10 hours produced amaurosis of

24 hours' duration without ophthalmoscopic signs suggest the possibility of cerebral rather than peripheral changes. The prognosis is good. Mydriasis (Hogg, Peterson, Cattani, Gatti) and hippus (Barabaschew) are recorded. Coloured vision has been recorded from sodium salicylate and other drugs (Hilbert).

Buss.—C. f. d. med. Wiss., xviii, 1875. Ries.—Berliner klin. Woch., 1875. Peterson.—Deutsche med. Woch., 1877. Gatti.—Gaz. degli Ospit. Milano, 1880. Knapp.—B. d. o. G., 1881. Gibson and Felkin.—Practitioner, 1889. Mann.—New York Med. Rec., 1892. de Schweinitz.—T. Am. O. S., 1895. Barabaschew.—La Clinique ophth., 1897. Snell.—T. O. S., xxi, 1901. Uhthoff.—In G.-S., xi, 2, 1901. Claiborne.—T. Am. O. S., 1905. Hilbert.—K. M. f. A., xlv, 1907.

Nitrobenzol and dinitrobenzol.—Nitrobenzol is the active constituent of artificial oil of bitter almonds and is a by-product of anilin dye manufactures. Dinitrobenzol affects workers with roburite and other explosives. The latter is the more poisonous. The ocular condition resembles that caused by quinine. In Nieden's and Snell's cases (dinitrobenzol) the field of vision was contracted, the retinal veins were distended and the arteries constricted. Bondi points out the difficulty of distinguishing between the arteries and veins except by difference of calibre. Changes in the blood like those of pernicious anæmia sometimes occur (Ehrlich and Lindenthal, Snell). Mydriasis and miosis have been described. The prognosis is good.

Filehne.—A. f. exp. Path. u. Pharm., ix, 1879. Litten.—Berliner klin. Woch., 1881. Nieden.—C. f. A., xii, 1888. Prosser White.—Practitioner, 1889. Dood.—Brit. Med. Jl., 1891. Hodson.—Lancet, 1891. Bondi.—Prager med. Woch., 1894. Snell.—Brit. Med. Jl., 1894. Pockley.—Austral. Med. Gaz., 1894. Ehrlich and Lindenthal.—Z. f. klin. Med., xxx, 1896. Mohr.—Deutsche med. Woch., 1902. Sesülinsky.—Mitt. a. d. Augenklinik in Jurjew, 1904.

Anilin.—Cases of amblyopia caused by anilin oil have been reported by Marchesi and Veasey. The latter found contraction of the fields, central scotomata, and slight papillitis. Litten noted violet coloration of the fundus in a case of anilin poisoning; the retinal vessels looked black, there were a few small hæmorrhages near the veins, but no amblyopia nor disturbance of colour vision. Discoloration, etc., of the conjunctiva and cornea with anilin and its derivatives occur (*cf.* Senn, Mellinghof).

Litten.—Berliner klin. Woch., 1881. McKinley.—T. O. S., vi, 1886. Silex.—A. f. A., xviii, 1888. Smith.—Lancet, 1894. Senn.—Correspondenzbl. f. Schweizer Aerzte, 1897. Veasey.—Am. Jl. of O., 1898. Marchesi.—Hygienekongress zu Como, 1899. Friedländer.—Neurol. Centralbl., 1900. Bocci.—Ann. di Ott., x, 1903. Berger.—A. f. A., l, 1904. Palermo.—Ann. di Ott., xxxiv, 1905. Senn.—Correspondenzbl. f. Schweizer Aerzte, 1897. Mellinghof.—K. M. f. A., xliv, 1906 (Bibliography).

Ergot.—Retinal anæmia (v. Bechterew), constriction of the retinal vessels (Alridge, Kortnew), retinal œdema, slight neuritis, etc., have been seen in ergot poisoning. No case of optic atrophy has been recorded. Slight transitory amblyopia and contraction of the field have been observed (v. Bechterew, Kortnew). Several cases of cataract ascribed to ergotism have been reported (Meyer, Tepljaschin, Schmidt-Rimpler, Logetschnikoff, and others). Experimental intravenous injection of the active principles of ergot produces passive

intra-ocular hyperæmia (Parsons). Nystagmus, hæmorrhage into the lids (Davidson), transitory exophthalmos (Tuczek), iritis, etc., have been described.

MEYER.—A. f. O., viii, 1862. ALRIDGE.—West Riding Lunatic Asylum Rep., i, 1871, 1872. TUCZEK.—A. f. Psych., xiii, 1882. HULME.—Med. News, 1887. v. BECHTEREW.—Neurol. Centralbl., 1892. KORTNEW.—In Nagel's Jahresbericht, 1892; A. d'O., xiii, 1893. SCHNEIDER.—Münchener med. Woch., 1902. ORLOW.—In Nagel's Jahresbericht, 1902.

Filix mas.—The visual disturbances caused by filix mas show resemblance to quinine amblyopia on the one hand and to lead poisoning on the other. Whether the toxic agent is filicic acid (Rulle, Poulson) or aspidin, and aspidinin (Walko) remains uncertain, and there is also great divergence of opinion as to the toxic dose of the drug. Bokai puts the latter as low as 4 grms., whilst Sidler-Huguenin found that 20—45 grms. might be innocuous amongst the workers in the St. Gotthard tunnel. Probably the general health of the patient is the determining factor. Katayama and Okamoto found ocular symptoms in 32·5 per cent. of cases of filix poisoning, and 35·7 per cent. in dogs: Maj found 2 cases of blindness amongst 70 people. Sidler-Huguenin in 78 cases found 12 deaths, 18 bilateral and 15 unilateral blindness, 4 bilateral and 1 unilateral permanent amblyopia, and 1 bilateral and 3 unilateral transient amblyopia. The amblyopia generally involves the whole field, thus accounting for the striking absence of details of the condition of the fields in the recorded cases. Quite a considerable proportion of the cases are unilateral, but too much stress must not be laid upon this fact, since the other seldom escapes entirely, especially in the early stages. I have seen a case in a young man who took a drachm of extract of filix mas three times a day for 10 days. One eye showed finally normal central vision with slight constriction of the field and temporal pallor of the disc, whilst the other showed complete blindness with total optic atrophy.

The chief ophthalmoscopic feature observed is extreme pallor of the disc with sharply defined edges. In transitory cases the ophthalmoscopic picture may be normal. In many cases the retinal vessels show abnormalities, especially constriction (Mikiji Yoda). Anatomically in experimental cases Masius and Mahaim found perivascular infiltration and breaking up of the myelin sheaths in the optic nerve, particularly in the neighbourhood of the optic foramen. Marked retinal changes—bright white spots, etc.—have been observed (Inouye), but it is known that filix mas may cause nephritis (v. Hofmann), and these changes may be secondary to this complication. No post-mortem examination in man has been reported, but there is a large literature of experimental observations on animals (Masius and Mahaim, Katayama and Okamoto, van Aubel, Nuel, Birch-Hirschfeld, de Schweinitz, and others). The most extensive changes are recorded by Nuel, but the most accurate are those of Birch-Hirschfeld, who, using the delicate Nissl method, found chromatolysis in the retinal ganglion cells and in the cells of the inner nuclear layer. Degenerative changes are found in the optic nerve, attributed by some to the degeneration of the ganglion cells, by others to the direct action of the poison. Masius and Mahaim again attribute the cellular changes

to defective nutrition following the vascular disorder.   In any case there can be little doubt that the lesion is essentially peripheral. There is only slight evidence of a specific action upon the sympathetic system (van Aubel, Sidler-Huguenin), but it is not disproved.

RULLE.—Dissertation, Dorpat, 1867. FRITZ.—C. f. A., xi, 1887. IMMERMANN.—Berliner klin. Woch., 1887. v. HOFMANN.—Wiener klin. Woch., 1890. POULSSON.—A. f. exp. Path. u. Pharm., xxix, 1892. KATAYAMA AND OKAMOTO.—Cei-I-Kwai Med. Jl., 1892; Viertels-jahrsschrift f. gerichtl. Med., 1894. GRANT.—Boston Med. Jl., 1893. MIKIJI YODA.—Vierteljahrsschrift f. gerichtl. Med., 1894. MASIUS.—Ann. d'Oc., cxiv, 1895. MASIUS AND MAHAIM.—Acad. roy. de Méd. de Belgique, 1896, 1898. INOUYE.—B. d. o. G., 1896. NUEL. —A. d'O., xvi, 1896; Internat. Med. Congress, Paris, 1900. DE SCHWEINITZ.—The Toxic Amblyopias, Philadelphia, 1896. SIDLER-HUGUENIN.—Correpondenzbl. f. Schweizer Aerzte, 1898. WALKO.—Deutsches Arch. f. klin. Med., lxiii, 1899. SIEGRIST.—A. f. A., xli, 1900. BIRCH-HIRSCHFELD.—A. f. O., l, 1900. UHTHOFF.—In G.-S., xi, 2, 1901 (Bibliography). NIEDEN.—Deutsche med. Woch., 1903. HABERKAMP.—Wochenschrift f. Hyg. des Auges, 1903. STUELP.—A. f. A., li, 1904. PALERMO.—Ann. di Ott., xxxiv, 1905. MEYER.— Schles. Gesellsch. f. vaterl. Kultur, 1905. v. KRÜDENER.—Z. f. A., xvi, Ergänzungshaft, 1906.

Lead.—The ocular complications produced by lead poisoning show great variety dependent upon the incidence of the lesion. They may be classified into peripheral, vascular, and cerebral lesions (Uhthoff) : moreover, lead may cause changes in other organs, such as the kidney, bringing about secondary disorders of vision.

Bilateral, sudden, more or less complete amaurosis is usually associated with little or no change in the fundus. Some of these cases are uræmic, accounting for the absence of anatomical signs post mortem (Tanquerel des Planches, Westphal), but the presence of nephritis does not necessarily entail this conclusion (Leber). Experimental observations have shown that lead may act directly upon the nervous system (Vulpian, Popow, Stieglitz, and others), and lead can be recovered from the brain as well as from other organs (Atkinson, Brailey, Bihler). This transient amaurosis is less common than the lead amblyopia dependent upon lesion of the peripheral visual tracts. Here the onset is gradual as a rule and may progress to complete amaurosis (in 10 per cent. of cases). The lesion of the optic nerve is inflammatory, very rarely primarily atrophic (Parisotti and Melotti). In 10 per cent. of cases the ophthalmoscopic examination is negative ; in 11 per cent. there is hyperæmia of the disc, in 30 per cent. papillitis, in 8 per cent. choked disc, in 12 per cent. neuroretinitis, in 29 per cent. partial or general postneuritic atrophy.   Changes in the retinal vessels are very frequent—constriction, white borders, etc. (Oeller, v. Schröder, Noyon, Folker, Hirschberg, Stood, and others).   Retinal hæmorrhages and white spots occur sometimes in the absence of albuminuric reti-nitis.   The syndrome of alcohol and tobacco amblyopia—centra. scotoma, temporal pallor of the disc, etc.—is relatively uncommon (Uhthoff) ; much more often there is concentric contraction of the field, frequently with irregular gaps at the periphery.   Ring scotoma (Landolt) is rare : temporal hemianopia has been reported once (Elschnig) : homonymous hemianopia (Westphal, Bihler, Hertel, and others) is indicative of encephalitis saturnina, occasionally of involvement of the optic tract (Bihler) with hemiparetic symptoms (v. Schröder, Hertel, Westphal).   Unilateral cases are rare (Oeller,

Noyon), but one eye may be attacked before the other (Hutchinson). Cases commencing with central scotoma often pass into a condition of grave amblyopia.

Anatomical observations have shown interstitial proliferation and thickening of the sheaths in the optic nerves (Brailey, Pflüger), hyaline degeneration of the vessels of the nerve, retina, and choroid (Oeller), papillitis (Westphal), etc. Changes in the brain and spinal cord are not infrequent, such as "hypertrophy" (Hitzig), chronic oedema (Kolisko), softening, hæmorrhages, etc. (Westphal and others). These lesions may cause ocular paralyses (v. Schröder, Snell, Mayer, Ring, Mannaberg, Zinken, Galezowski—sixth nerve; Wood, Mannaberg, Landesberg, Lagleyze—third nerve; Chvistek, Bach, Wadsworth, Galezowski—third and sixth nerves). Experimental efforts have failed to produce visual disturbance, though large doses have been administered (0·1—0·5 grm. of lead chloride *per diem* to dogs, Combemale and François), and paralyses are rare (Stieglitz, Schaffer, Rybakoff), though changes are described in the cells of the anterior horns.

Beer.—Lehre v. d. Augenkrankheiten, ii, 1817. Tanquerel des Planches.—Traité des Maladies de Plomb, Paris, 1839. Hutchinson.—R. L. O. H. Rep., vi, 1867; vii, 1871; ix, 1879. Schneller.—K. M. f. A., ix, 1871. Lunn.—Med. Times and Gaz., 1872. Samelsohn.—K. M. f. A., xi, 1873. Brailey.—R. L. O. H. Rep., viii, 1876. Reid.—Ann. d'Oc., lxxvii, 1877. Galezowski.—Rec. d'O., 1877, 1878. Atkinson.—Lancet, 1878. Hirschberg.—A. f. A., viii, 1878; Berliner klin. Woch., 1883. Landolt.—Ann. d'Oc., lxxxiii, 1880. Stephen Mackenzie.—T. O. S., i, 1881. Oeller.—Virchow's Archiv, lxxxvi, 1881. Wadsworth.—T. Am. O. S., 1881; Boston Med. and Surg. Jl., 1885. Maier.—Virchow's Archiv, xc, 1882. Pflüger.—Univ.-Augenklinik in Bern, 1883. Popow. —Virchow's Archiv, xciii, 1883. Stood.—A. f. O., xxx, 3, 1884. Oliver.—Brit. Med. Jl., 1885, 1891. Parisotti and Melotti.—Rec. d'O., 1885. v. Schröder.—A. f. O., xxxi, 1, 1885. Rampoldi.—Ann. di Ott., xvi, 1887. Uhthoff.—A. f. O., xxxiii, 1, 1887; in G.-S., xi, 2, 1901. Westphal.—A. f. Psych., xix, 1888. Günzburg.—A. f. A., xx, 1889. Buzzard. —Brain, xiii, 1890. Combemale and François.—Comptes rendus, cxi, 1890. Hertel.— Charité Ann., xv, 1890. Jeaffreson.—Brit. Med. Jl., 1890. Stieglitz.—A. f. Psych., xxiv, 1892. Bach.—A. f. A., xxvi, 1893. Ring, Cram and Miller.—Am. Jl. of Med. Sc., 1896. Wood.—Am. Med. News, 1897. Elschnig.—Wiener med. Woch., 1898. Noyon.—Ann. d'Oc., cxix, 1898. de Schweinitz.—Ophth. Rec., 1898. Taylor.—Lancet, 1898. Folker.—T. O. S., xix, 1899. Bihler.—A. f. A., xl, 1900. Dodd.—T. O. S., xx, 1900. Snell.—T. O. S., xxiv, 1904. Ogg.—Clin. Jl., 1905.

Diseases of the Nervous System.

The ophthalmic complications of diseases of the nervous system form too large a subject to receive exhaustive treatment in this work. Only the purely ocular complications will be considered. For further details the reader is referred to special monographs, particularly the recent work of Uhthoff.

*Uhthoff.—In G.-S., xi, 2, 1904-

Diseases of the Spinal Cord.

Tabes dorsalis.—Optic atrophy occurs in about 10—20 per cent. of cases of locomotor ataxia; statistics derived from ophthalmic clinics are naturally too high—40 per cent. (Uhthoff). It is inadvisable to lay too much stress upon these statistics since optic atrophy may be

the first sign of tabes, and the appearance of other and indisputable evidence of the disease may be long delayed (2 years, Berger; 7 years, Hoffmann and Bernhardt; 10 years, Charcot; etc.) The incidence of tabes and therewith of tabetic atrophy increases with age, reaching the highest point between 30 and 50. Tabetic atrophy is about twice as common in men as in women. It is commonest in the preataxic stage—preataxic 29 cases, ataxic 12, paralytic 3, indefinite 10 (Berger); in 165 tabetics optic atrophy 55, of whom 15 (27·2 per cent.) were ataxic (Mann); in 58 ataxic tabetics optic atrophy 6 (10 per cent.) (O. Foerster). The converse proposition, that optic atrophy has a good influence over the other tabetic symptoms (Dejerine, Spiller, O. Foerster, Bürstenbinder), is not supported by the evidence.
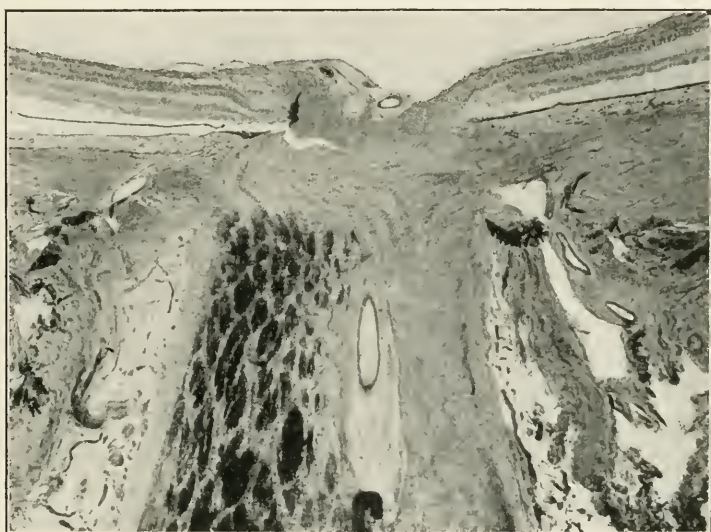
FIG. 844.—TABETIC ATROPHY.

From a specimen by Wintersteiner, photograph by Coats. Stained by Weigert-Pal method.

Ophthalmoscopically tabetic atrophy is characterised by the sharp definition of the edge of the disc. The disc itself is chalky white, or greyish or bluish-white: Uhthoff regards a greenish-white disc as more characteristic of secondary atrophy and of differential diagnostic significance. Pallor of the disc may precede failure of vision by a considerable period, never the reverse: the appearance of the disc is no sure guide to the deficiency in visual perceptions. The retinal vessels are little altered at first, but later undergo constriction, though never to the extent which occurs in postneuritic atrophy. White lines along the vessels are rare. Atrophic cupping of the disc depends upon the amount of previous physiological cupping. The occurrence of papillitis (Wilbrand, Oliver, and others) must be regarded as an epiphenomenon, dependent upon coincident lues, retrobulbar neuritis,

etc. Similarly, temporal pallor is inconsequential. Retinal and choroidal changes are usually attributable to syphilis.

The general consensus of opinion as to the pathological anatomy of the optic atrophy of tabes is that the primary seat of the disease is in the retina, commencing in the ganglion cells and nerve-fibre layer (Leber, Popow, Wagenmann, v. Grosz, Coppez, v. Leyden and Goldscheider, Elschnig, Uhthoff, and others). In the nerve the atrophy is generally most marked near the globe, diminishing towards the cerebrum : changes in the chiasma and tracts are absent in the early, and are slight even in the later stages. The visual tracts above the so-called primary optic centres are not affected. In a case of Wagenmann's there were medullated nerve-fibres in the retina which lost their sheaths in the early stage of tabetic optic atrophy.

The degeneration of the nerve-fibres is therefore generally regarded as secondary to disease of the ganglion cells, though in some cases the fibres may themselves be primarily attacked (Uhthoff). The changes in the neuroglia are secondary, few authors considering them the original seat of the disease (Basso). Buzzard, v. Leyden and Goldscheider, and others attribute the disease to primary affection of the blood-vessels or of the sympathetic nerve, but anatomical investigations do not bear out these theories.

Degeneration of the nerve-fibres commences with breaking up of the myelin sheaths. Later the axis cylinders become varicose and degenerate. A few granular cells (Fettkörnchenzellen) may make their appearance (Leber), but as a rule they are entirely absent. The normal fibres, which stain by Weigert's method, gold chloride, osmic acid, etc., are readily distinguished from the degenerating fibres, which stain with carmin, nigrosin, Marchi method, etc. There is no proliferation of neuroglia in the early stages : indeed, the finer processes of the interstitial connective tissue undergo atrophy ; these, however, belong probably to the mesoblastic pial septa. The shrinking of the atrophic nerve-fibres may leave small spaces which are traversed by neuroglial processes. Soon the neuroglia shows increase of nuclei and thickening of the processes, so that in the later stages the nerve is largely replaced by thickened septa and their offshoots. The longer the atrophy has lasted the greater the changes in the interstitial tissues, though even then there is a conspicuous absence of active proliferation.

The retina shows marked degenerative changes in the ganglion cells and in the nerve-fibre layer, without any signs of inflammation or proliferation. Both in the retina and nerve the vessels exhibit degenerative changes in the walls, chiefly hyaline thickening, but these occur chiefly in the later stages and may be altogether independent of the tabetic affection. The exact relationship between the ganglionic disease and the nerve-fibre degeneration, though probably that indicated above, has been the subject of discussion similar to that surrounding the disease of the spinal ganglia and the degeneration of the posterior columns of the cord (cf. Moxter, Wollenberg, and others) : the two conditions are not, however, strictly analogous.

The vulnerability of the optic nerve in tabes has been explained on various theories, none of which are wholly satisfactory. Wharton

Jones attributed it to the sympathetic nerve on the analogy of amaurosis occurring in spinal injuries; Mooren, Rieger, v. Forster and Berger to reflex vascular changes induced by lesion of the medulla oblongata; Allbutt, Weiss, and others to chronic ascending meningitis; Mauthner to descending atrophy originating in the primary optic centres.

ROMBERG.—Lehrbuch der Nervenkrankheiten, 3rd Edit., 1857. DUCHENNE.—Arch. gén. de Med., 1858. v. GRAEFE.—K. M. f. A., iii, 1865. LEBER.—A. f. O., xv, 3, 1869; in G.-S., v, 1877. BERGER.—A. f. A., xix, 1888. MANN.—Allg. med. Centralzeitung, 1902. O. FOERSTER.—Monatsschrift f. Psych. viii, 1900. DEJERINE AND THOMAS.—Traité des Maladies de la Moelle épinière, 1902. SPILLER.—Philadelphia Med. Jl., 1898. WILBRAND. —K. M. f. A., xxxix, 1901. OLIVER.—T. Am. O. S., 1900. POPOW.—Deutsche Z. f. Nervenheilk., iv, 1893. WAGENMANN.—A. f. O., xii, 4, 1894. v. GROSZ.—Z. f. A., ii, 1899, Beilageheft. v. LEYDEN AND GOLDSCHEIDER.—In Nothnagel, 1897. ELSCHNIG.—Wiener klin. Woch., 1899. BUZZARD.—Brain, 1884. GLIKSMANN.—Dissertation, Freiburg, 1900. *UHTHOFF.—A. f. O., xxxii, 3, 1886; A. f. Psych., xxi, 1890; in G.-S., xi, 2, 1904 (Bibliography). MARBURG.—Wiener klin. Woch., 1903. MOTT.—Arch. of Neurol., ii, 1903. LÉRI. —Nouvelle Iconographie de la Salpétrière, xvii, 1904. MARIE.—Rec. d'O., 1904. NICOLAI. —Charité Annalen, xxviii, 1904. GALEZOWSKI AND LOBEL.—Rec. d'O., 1906. SPIELMEYER. K. M. f. A., xliv, 1906.

Combined sclerosis.—In combined sclerosis of the posterior and lateral columns of the cord simple progressive optic atrophy, having all the features of tabetic atrophy, occurs (Westphal, Gowers, Oppenheim, and others).

WESTPHAL —A. f. Psych., viii, ix, 1879. GOWERS.—Lancet, 1886. OPPENHEIM.— Neurol. Centralbl., 1888. UHTHOFF.—In G.-S., xi, 2, 1904 (Bibliography).

Myelitis.—In 1879 Erb and Steffan reported a case of dorsal transverse myelitis with retrobulbar neuritis and slight papillitis. About fifty cases of optic neuritis associated with myelitis have now been published (Noyes, Sharkey and Lawford, Achard and Guinon, Kalt, Elschnig, Schanz, Katz, Dalén, Frederick Taylor, Bielschowsky, James Taylor and Collier, Hillion). In some cases the picture of a pronounced choked disc was present (Chisholm, Knapp, Dreschfeld, Mahokian, Henneberg): in others visual disturbance without ophthalmoscopic signs points to retrobulbar neuritis. Primary optic atrophy is rare (Girandeau, Singer), as also partial atrophy affecting the temporal side of the disc (Peltesohn). The retina is usually free from abnormality (*cf.* Katz, Elschnig). The visual disturbance generally amounts to complete amaurosis, which is commonly transient but eventuates in permanent amblyopia. There is often an interval, usually of a few days only, between the affection of the two eyes (Noyes, Elschnig). Pain on movement of the eyes and other signs of retrobulbar neuritis may be present (Chisholm, Schanz, Bielschowsky, and others). The optic affection may precede signs of the myelitis by days or months (Erb and Steffan, Knapp, Dalén, and others), usually only a short period. The symptoms may be simultaneous (Noyes, Rumf, Dreschfeld); rarely those of myelitis precede those of optic neuritis (Seguin). The field of vision varies greatly in different cases—temporal hemianopia (Noyes, Steffan, Schanz), central scotoma (Noyes, Steffan, Elschnig, Schlüter, Friedmann), concentric contraction (Katz, Elschnig, Schanz, etc.).

Anatomical investigations have been possible in a considerable

number of cases (Knapp, Achard and Guinon, Dreschfeld, Sharkey and Lawford, Kalt, Elschnig, Hoffmann, Katz, Mager, Dalén, Bielschowsky, and others). The optic nerves show changes analogous to those in the cord—softening, degeneration of the fibres, neuroglial proliferation, etc. In most cases the optic nerves, chiasma, and tracts have all been involved (Sharkey and Lawford, Kalt, Knapp, Elschnig, Katz, Dalén, Bielschowsky); occasionally only the optic nerves (Dreschfeld). Isolated affection of the chiasma or tracts does not occur. The inflammatory nature of the process is admitted by all authors : infiltration and thickening of the septa, vascular disease, inflammation of the sheaths, etc., are frequently reported, and degeneration of the nerve-fibres is regarded as secondary (Sharkey and Lawford, and others). Bielschowsky considers the neuritis parenchymatous and primary.

The pathogenesis of the disease offers peculiar difficulties. There can be no doubt that the myelitis and optic neuritis depend upon the same underlying cause (Erb). Anatomical continuity of the inflammatory process from the cord to the optic nerves cannot be demonstrated ; indeed there may be lumbar or dorsal myelitis with complete escape of the cervical region of the cord. Transference of toxins by the blood stream seems the most probable explanation. The suggestion that the sympathetic system is at fault is purely theoretical, and is not supported by satisfactory evidence. There are no signs of general meningitis and the other cranial nerves escape.

Erb.—A. f. Psych., x, 1880. Steffan.—B. d. o. G., 1879. Seguin.—Jl. of Nerv. and Mental Dis., Chicago, 1880. Noyes.—A. f. A., x, 1881. Chisholm.—A. of O., xi, 1882. Dreschfeld.—Lancet, 1882; Brit. Med. Jl., 1894. Coxwell.—Brain, 1883. Sharkey and Lawford.—T. O. S., iv, 1884. Peltesohn.—C. f. A., x, 1886. Achard and Guinon.— A. de Méd. exp., 1889. Kalt.—Ann. d'Oc., cii, 1889. Elschnig.—A. f. A., xxvi, 1893. Schanz.—Deutsche med. Woch., 1893. Hoffmann.—Neurol. Centralbl., 1894; A. f. Psych., xxxviii, 1896. Katz.—A. f. O., xlii, 1, 1896. Buzzard and Risien Russell.—Brit. Med. Jl., 1898. Dalén.—A. f. O., xlviii, 3, 1889. Frederick Taylor.—Guy's Hosp. Rep., 1899. Bielschowsky.—Myelitis u. Sehnervenentzündung, Berlin, 1900. Taylor and Collier.—Brain, 1901. Hillion.—Thèse, Paris, 1907. Brissaud and Brécy.—Rev. neurologique, 1904. Uhthoff.—In. G.-S., xi, 2, 1904.

Multiple or disseminated sclerosis.—Charcot and his pupils first drew attention to the ocular defects associated with multiple sclerosis. Since their time a vast literature has grown around the subject, including the names of many prominent neurologists and ophthalmologists. Uhthoff in 100 cases found the fundi normal and no visual disturbance in 48 per cent. There was marked optic atrophy in 3 per cent., incomplete atrophy in 19 per cent., temporal pallor of the disc in 18 per cent., optic neuritis in 5 per cent. The optic nerves are affected therefore much more commonly than in tabes, more often indeed than in any other disease of the nervous system except cerebral tumour (Uhthoff). The clinical features are essentially those of retrobulbar neuritis—central scotoma without or with contraction of the peripheral field, regular concentric contraction, ring scotoma, etc.: hemianopic fields are rare. Choked disc has been reported by Uhthoff, Bruns and Stölting, and Kampherstein.

The pathological anatomy of the optic nerves in insular sclerosis is

characterised by significant differences in detail from that in other diseases, differences in accord with those found in similar lesions in

Fig. 845.—Disseminated Sclerosis.
Specimen by Gordon Holmes, photograph by Coats. Chiasma, optic nerves, and tracts. Stained by Weigert-Pal method, the light areas being patches of degeneration.
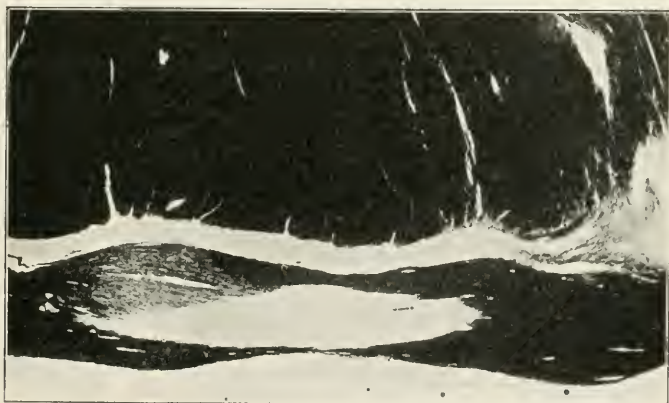
Fig. 846.—Disseminated Sclerosis.
Specimen by Gordon Holmes, photograph by Coats. Patch of sclerosis in optic tract. Stained by Weigert-Pal method.

other parts of the central nervous system.   It resembles tabetic atrophy in the absence of pronounced signs of inflammation; it differs

from it in the prolonged escape in great part of the axis cylinders in spite of their denudation by destruction of the medullary sheaths. Whereas in the former condition extensive secondary degeneration of the nerve-fibres, extending far beyond the site of most pronounced disease, occurs, in disseminated sclerosis there may be little or no degeneration outside the area of the lesion. Clinical observations, indeed, show that conduction may be effectual, though disordered, through the actual lesion, the defect appearing to be essentially one of insulation (Gowers). The medullary sheaths show even greater degeneration than in correspondingly early stages of tabes, whilst the axis cylinders may show no change or only slight varicosities. There is cholin in the blood (Tredgold). These changes are more readily demonstrated in parts of the nervous system, where the nerve-fibres are larger than in the optic nerves, but they are quite definite in this position. In the later stages the picture of primary atrophy is more nearly simulated, and it may be impossible to distinguish between the two conditions. Special methods of staining (carmine, hæmatoxylin, Weigert's alum carmine, etc.) display the nuclei in such a manner as to show that there is extensive proliferation of the finer strands of interstitial tissue derived from the pial and glial septa (Uhthoff). The shrinking of the sclerosed part of the nerve is often much more marked than in tabetic atrophy, and this is attributable to an active process of proliferation in the interstitial tissues (Uhthoff). The increase in the number of nuclei, the cellular infiltration, etc., tend to show that the process is inflammatory, though these signs are confined to the larger septa and are in no degree conspicuous. All signs of inflammation may indeed fail, and these cases afford the greatest support to the theory of a parenchymatous lesion (Thomas and Dejerine, and others), though the majority of cases militate against this view.

The comparative absence of degeneration of the nerve-fibres outside the affected area is paralleled by the absence of changes in the ganglion cells of the retina, which may show no changes, at any rate by ordinary methods of investigation, in spite of extensive changes in the nerve.

The ganglion cells throughout the nervous system show an absence of acute change; they nearly always contain a large amount of granular pigment, and in the later stages of the disease the cells undergo a chronic atrophy which is probably secondary to the destruction of their axons (Tredgold).

The blood-vessels are generally altered, often profoundly, but the inconstancy of these changes and their distribution when present militate against the theory of a vascular pathogenesis for the disease (Rindfleisch, and others). Thus, the vascular changes, chiefly in the form of increase in the number and widening of the lumen of the finer vessels, often occur in otherwise healthy spots in the nerve, whilst they may be entirely absent in the sites of grosser lesions.

The pathological conditions are suggestive of the presence of a circulating toxin as the cause of the disease, but there is at present no proof that such is the case.

Noyes.—Arch. of Sc., 1873. Nettleship.—R. L. O. H. Rep., ix, 1877; T. O. S., iii, 1883. Eales.—T. O. S., iv, 1884. Sharkey.—T. O. S., iii, 1883. Schöler and Uhthoff.

—Beiträge zu den Sehnerven-u. Netzhauterkrankungen bei Allgemeinleiden, Berlin, 1885. PELTESOHN.—C. f. A., x, 1886. HUGHLINGS JACKSON.—T. O. S., vii, 1887. SWANZY.— T. O. S., viii, 1888. HABERSHON.—T. O. S., ix, 1889. UHTHOFF.—A. f. Psych., xxi, 1889; in G.-S., xi, 2, 1904; Ophthalmoscope, 1905. ZIMMERMANN.—A. of O., xx, 1891. CHARCOT. —A. d'O., xiii, 1893. BUZZARD.—Brit. Med. Jl., 1893; 1899; Lancet, 1896; 1897. LÖBBERT. —A. f. Psych., xxix, 1897. DISCUSSION ON RETRO-OCULAR NEURITIS.—T. O. S., xvii, 1897. GOLDSCHEIDER.—Z. f. klin. Med., xxx, 1898. SCHUSTER AND BIELSCHOWSKY.—Z. f. klin. Med., xxxiv, 1898. DE BONO.—Arch. di Ott., vi, 1899. ELSCHNIG.—Wiener klin. Woch., 1899. BRUNS AND STÖLTING.—Z. f. A., iii, 1900. TAYLOR.—Brit. Med. Jl., 1902, KAMPHERSTEIN.—A. f. A., xlix, 1903. ROSENFELD.—Neurol. Centralbl., 1903. BIEL-SCHOWSKY.—Neurol. Centralbl., 1903; 1904. BIELSCHOWSKY AND POLLACK, STRÄHUBER.— Neurol. Centralbl., 1904. SPILLER AND CAMP, WEBBER.—Jl. of Nerv. Dis., 1904. TREDGOLD. —Rev. of Neurol. and Psych., ii, 1904. BERGER.—Jahrbuch f. Psych., xxv, 1905. E. MÜLLER. —A. f. Psych., xl, 1905. FLEISCHER.—Ophth. Klinik, x, 1906. ORMEROD.—Brain, xxx, 1907.

Syringomyelia.—The purely ocular complications of syringomyelia are rare compared with alterations in the pupils, paralyses of extrinsic muscles, nystagmus, etc. Optic atrophy and papillitis occur, but are to be regarded as epiphenomena dependent upon coincident tabes or progressive paralysis, etc. Concentric contraction of the field with normal fundus has been frequently described.

UHTHOFF.—In G.-S., xi, 2, 1904 (Bibliography).

Tumours of the cord.—Papillitis and optic atrophy (Ormerod and Hadden, Schlagenhaufer) may occur from intra-cranial complications or from direct extension of diffuse sarcoma (Ormerod and Hadden), etc. Rarely optic neuritis may arise apparently in the same manner as in myelitis (Schultze, Nonne, Heubner).

SCHULTZE.—A. f. Psych., viii, 1879. ORMEROD AND HADDEN.—Brit. Med. Jl., 1887. NONNE.—A. f. Psych., xxxiii, 1900. HEUBNER.—A. f. Psych., xxxiv, 1901.

Injuries of the cord.—Amblyopia and amaurosis following, some-times at a long interval, injuries of the cord are described (Wharton Jones, Allbutt, Mooren, Nieden, and others).

WHARTON JONES.—Brit. Med. Jl., 1869. ALLBUTT.—Lancet, 1870. MOOREN.—Ophth. Mittheil., Berlin, 1874. NIEDEN.—A. f. A. xii, 1883. STERLING.—Jl. of Ophth., Otol., and Laryngology, ii, 1890. FOWLER.—Jl. of Ophth., etc., 1891.

DISEASES OF THE BRAIN.

The ocular complications of diseases of the brain belong rather to the domain of neurology than ophthalmology, and are beyond the scope of this work. One important exception, the optic neuritis associated with intra-cranial disease, must be made.

*WILBRAND AND SAENGER.—Die Neurologie des Auges, Wiesbaden, 1900— . PARSONS.—The Neurology of Vision, London, 1904.

Papillœdema ("choked disc," papillitis, optic neuritis).—Some distinction in nomenclature is needed between the papillitis of moderate degree due to a great variety of causes and the papillitis with pronounced swelling—of more than 2 D—associated with increased intra-cranial pressure. I suggest the use of the term "papillœdema" to replace " choked disc " (Stauungspapille).

In the varied causes of papillœdema the feature which assumes

paramount importance is increase of intra-cranial pressure. Intra-cranial pressure, like intra-ocular pressure, is dependent upon intra-vascular pressure. It is necessary therefore to review briefly our knowledge of the conditions which control and modify the intra-cranial circulation and of the relations which subsist between the intra-cranial and intra-ocular circulations.

Mention has already been made of the fact that possibly an undue amount of importance has been attached to the conditions of the retinal circulation as an index to those of the intra-cranial vessels. As has been pointed out, the major vascular supply of the eye in lower mammals is derived from the external carotid artery, *i. e.* from a purely extra-cranial source, though the intimacy of relationship with the internal carotid in its intra-cranial course, which obtains in the higher primates, reaching its acme in man, is foreshadowed by minor anastomoses even in the inferior species (Vol. III, Chap. XVII). Morphology, therefore, affords a strong argument which we have already seen put to the crucial test of experimental proof, against the overwhelming importance of intra-cranial conditions as affecting the ocular circulation. It is impossible, however, on anatomical grounds alone to suppose that this view can be completely maintained for man and the higher primates.

Previous experiments upon this subject have been extremely few in number, and have led to no definite conclusion. Only those of v. Schultén require passing mention. Indeed, it is only since the exhaustive researches of Leonard Hill upon the cerebral circulation, which have demonstrated the accuracy of the Monro-Kellie doctrine, that the time has become ripe for the investigation. We now know the very small part which is played in the cerebral circulation by the cerebro-spinal fluid—that this fluid normally exists inside the skull in such small quantities that its ebb and flow are totally inadequate to counteract the larger changes of volume induced by experimental procedures in the intra-cranial vessels. At the same time it is quite possible that extremely small vasomotor changes over a very limited area, *e. g.* over one or more of the nerve nuclei in the medulla oblongata, might be so counteracted, and yet be accompanied by physiological results of profound importance, although the actual changes in volume might be so minute as to entirely elude the comparatively gross methods of observation. The demonstration of nerve-fibrils upon the cerebral vessels—fibrils whose destination can hardly be other than the muscle walls of the vessels—must be regarded as strong evidence in favour of this view. As regards the larger variations, the correctness of the Monro-Kellie doctrine is placed beyond cavil, and so far we may consider the quantity of blood inside the cranium to be constant.

It follows from this law that, with the possible exceptions indicated above, all changes in the general blood-pressure produce only passive changes in the cerebral circulation. Unlike what happens in other parts of the body, these changes are not manifested as changes in the volume—dilatation or constriction—of the vessels, but as variations in the rate of flow of the blood. Thus, if the aortic pressure rises, as *e. g.*

by constriction of the vessels of the splanchnic area, the vena cava pressure remaining constant, there will be an increased velocity of flow through the brain.   The increased pressure in the arterioles cannot be relieved by dilatation, but the blood is hurried on.

Let us suppose for a moment that the ocular circulation is a direct offshoot from the intra-cranial circulation, the ophthalmic artery forming the connecting link between the two systems.   When the blood reaches the orbit, one of two things may occur—the ophthalmic artery may dilate passively, the flow being again retarded to the normal rate, or it may remain constant, the increased velocity continuing.   There is no evidence that the larger arteries can accommodate themselves— at any rate to the required extent—in the manner suggested by the first alternative, their function being to transmit the blood with as little loss of pressure and velocity as is consistent with friction and other physical conditions.   On reaching the eye, the vessels again become shut up in a closed box.   If this were rigid, like the skull, the same conditions would obtain, and increased velocity of flow would be the only result of a rise in general blood-pressure, unless, indeed, dilatation of the vessels occurred at the expense of the intra-ocular fluid. There is no evidence that filtration from the eye varies thus with the rapid changes in blood-pressure, but there is evidence that the globe is not rigid, and that the vessels do dilate.   Consequently the results which we have already obtained are not inconsistent with the view that the ocular circulation is a direct offshoot from the intra-cranial, but on the other hand they are equally consistent with the view that the ocular circulation is a mere offshoot from the general extra-cranial circulation ; for as is well known, when vaso-constriction occurs in a large area of the body, notably in the splanchnic area, the large increase in general blood-pressure thereby induced leads to the passive dilatation of other—for the time being—less important areas, so that accommodation is thus afforded to the displaced blood.

A rise in general venous pressure, with coincident constant or diminished arterial pressure, will produce diametrically opposite results, viz. retardation of the cerebral blood flow, with passive constriction of the intra-ocular arterioles, and that irrespective of the rich anastomosis of the extra-ocular veins with the facial, pterygoid, and other veins of the extra-cranial system, which obtains in all mammals, whether their arterial ocular supply is mainly extra- or intra-cranial.

Under normal intra-cranial conditions, therefore, it would seem to be immaterial, physiologically, whether the ocular circulation is derived from intra- or extra-cranial sources, or both.   If, however, the arterial supply is wholly or mainly of intra-cranial origin, as in man, or in the dog with ligatured external carotid, whilst the ocular veins communicate freely with both the intra- and extra-cranial systems, it is clear that intra-cranial complications may be expected to affect profoundly the ocular circulation, both from the arterial side and also to a less extent from the venous.   Any intra-cranial condition which impedes the flow of blood in the internal carotid may be expected to lead to passive constriction of the intra-ocular arterioles ; but such intra-cranial conditions usually affect the venous sinuses even more profoundly, leading to

partial blockage, with coincident slowing of the circulation. This latter effect, however, will not necessarily lead to increased ocular venous pressure, owing to the free communication with the general system of veins, which themselves anastomose so freely as to tend rapidly to the equalisation of pressures. There are, besides, physiological complications to which it will be necessary to draw attention now.

Two physical conditions—that the skull is an hermetically sealed box, and that the quantity of cerebro-spinal fluid is unimportant, except as a lubricant—have already been mentioned. To these a third must be added—the fact that the brain substance is incompressible, though the brain as a whole, with its contained vascular channels, is as compressible as a sponge. It follows from these conditions, that under normal circumstances, with the skull intact, the intra-cranial pressure is already raised, and that its amount is equal to the coincident cerebral capillary pressure. When the skull is freely opened the intra-cranial pressure falls to the level of the atmospheric pressure. When the intra-cranial pressure is artificially raised by the introduction into the skull of a foreign body of known volume, two results follow: (1) by virtue of its compressibility as a vascular network, blood is squeezed out of a certain portion of the brain; (2) by virtue of its incompressibility *quâ* brain-substance, it is bodily dislocated slightly, and the pressure is transmitted to other parts. Now the pressure is not transmitted equally in all directions, a fact which is due chiefly to the resistence, partly of the falx cerebri, but principally of the tentorium cerebelli, which seems to be architecturally designed to protect the cerebellum, and through it the pons and medulla, from pressure.

The intra-cranial tension can be raised by other means, such as the injection of an innocuous fluid. In order to keep it raised and constant the injection must be continuous, as the fluid is rapidly absorbed into the venous sinuses. The actual amount absorbed during an ordinary experiment is small, and is not sufficient to embarrass the right heart, as has been suggested. By this method, when applied with moderate rapidity in the parietal region, the brain is first forced down towards the isthmus tentorii and foramen magnum, which are effectually plugged. The pressure is then nearly equally distributed over the surface of the brain, which does not happen by the method of local compression.

A combination of the two methods is obtained by bleeding the animal from one carotid into its skull. In this experiment the pressure is at first one of fluid pressure, though probably never so equally distributed as with saline solution; later the blood coagulates, and acts as a foreign body, taking up a definite space, the serum alone being capable of rapid absorption. The clot then produces a condition of local compression.

Since an accurate knowledge of the effects of increased intra-cranial pressure upon the cerebral circulation form the basis of a correct interpretation of the results obtained in the ocular circulation, it is necessary to describe them somewhat fully. The first effect of a rise of tension by the application of fluid pressure is venous stasis. This

can be observed by watching the small vessels of the pia mater—magnified, if necessary—through a glass window let into the skull. The skull must, of course, be completely closed, so as to avoid the errors of many previous observers. If the window includes the longitudinal sinus, it will be seen that this gradually becomes narrower as the pressure rises, and may even be completely obliterated (Cushing). The venous stasis leads to irritation of the bulbar centres, but only when the pressure is high, and is approaching the arterial tension, for the metabolism of the brain is very slow, so that the vital centres are protected. They are specially protected from local pressure applied to other parts of the brain by the notable pressure discontinuity, the blocking of the isthmus tentorii by the translocation downwards of the cerebrum, and by the partial escape of the medulla in some animals below the level of the foramen magnum. When, however, the fluid pressure is high, the cardio-inhibitory and respiratory centres are stimulated—the heart is slowed and may be stopped, even fatally, through the vagi. Mechanical compression of the vagi against the bone may be a factor in producing this result; in any case it is avoided by section of these nerves. The vaso-motor centre is also stimulated. If the cortex is inspected through a window in the skull, the circulation is seen to stop when the pressure rises to the arterial tension. The brain becomes pale; the veins in the sulci remain filled with stagnant blood, and the larger arterioles pulsate, but the capillary circulation has ceased. The animal, however, does not die. What is seen to occur in the cortex also occurs in the medulla. The vasomotor centre is asphyxiated, and thereby stimulated. The vessels of the splanchnic area contract, the blood-pressure rises, the blood is forced on, and the circulation is re-established. This process can be repeated over and over again, until the pressure—intra-cranial and arterial—may be raised as high as 250 mm. Hg. Eventually the vasomotor apparatus is paralysed, and the animal dies. If the spinal cord is cut high up, or the medulla cocainised, no rise of blood-tension accompanies the increase of intra-cranial pressure; the response, too, is feeble if the animal is in a bad condition.

From experiments which I have made local pressure applied for a short time to the parietal region appears to be without effect upon the intra-ocular circulation, but fluid pressure is more equally distributed and may have a very definite influence. It seems clear that, at any rate in the dog, the arteries at the base of the skull are very well protected from pressure, especially the transmission of pressure applied at a distance; and that any effect upon the venous sinuses is counteracted by the free anastomosis of the ocular veins with those of the general circulation. It is practically impossible to tie off the anastomoses, but valuable results, both with regard to the normal ocular circulation and to its relationship with the intra-cranial circulation, would result from any method whereby the ocular venous pressure could be measured. It is in this direction, and in experiments upon monkeys, that further investigation is required.

The chief cause of papilloedema is the presence within the cranium

of some adventitious material, which may be solid or fluid. To the former category belong malignant growths, inflammatory deposits, and blood-clots; to the latter distension of the lymph spaces and reservoirs by an abnormal amount of cerebro-spinal fluid, which may also be abnormal in constitution as the result of meningeal inflammation, etc. Clinically the form of adventitious deposit which most frequently gives rise to choked disc is tumour of the brain. Other causes, more or less in order of frequency, are syphilis, tuberculosis, brain abscess, meningitis and hydrocephalus. Rarer causes are anæmia, nephritis, sinus thrombosis, cysticercus, deformities of the skull, etc.

Brain tumour.—According to Gowers papillœdema occurs in at least four fifths of all cases of brain tumour. Annuske and Reich found it recorded in 95 per cent. of cases collected from the literature (88 cases). Oppenheim found it in 90 per cent of cases (100 cases). It was absent in only 5 cases out of 31 examined post-mortem by Bruns, viz. in a small angioma of the central convolutions, a gumma of the occipital lobe, a glioma pontis, and 2 cases of cysticercus of the fourth ventricle. It occurs most often in tumours of the cerebellum and base of the brain: it is less frequently present in tumours of convexity of the cerebral hemispheres. Edmunds and Lawford found papillœdema out of a total of 107 cases in 20 out of 23 cases of cerebellar tumour, in 74 per cent. of tumours of the base, and in 50 per cent. of tumours of the convexity. The relative freedom of cases of tumours affecting the convexity of the brain is doubtless due to the protection of the basal parts from pressure afforded by the dense tentorium cerebelli (*v.* p. 1352) —a point emphasised by Gròsz.

Papillœdema is an early sign in tumours of the cerebellum and base. It is late in development in tumours of the cerebrum, especially of the frontal convolutions (Bruns). In 23 cases of cerebral tumour collected by Oppenheim ophthalmoscopic changes were present in 82 per cent., typical choked disc in 65 per cent.. papillitis in 5 per cent., hyperæmia in one case. In 55 cases of tumour of the frontal lobes Williamson found papillœdema in 64 per cent. Gowers reports a case of Barlow's in which a small tumour of the medulla oblongata caused choked disc, but tumours in this situation more often cause death too rapidly for its development.

Kampherstein has collected 72 cases of brain tumour with papill- œdema: of these 23 affected the cerebellum (32 per cent.), 12 the frontal lobes, 6 the temporal, 4 the central convolutions, 5 the occipital, 2 the epiphysis, 2 the third ventricle, 3 the corpus callosum, 2 the optic thalamus, 7 the colliculi, 3 the hypophysis, 1 the fourth ventricle, 2 the cerebellar peduncles.

Unilateral choked disc in brain tumour is rare (Gowers 3 cases, Hughlings Jackson 2 cases, Field, Ulrich). The tumour is generally situated near or extends to the chiasma or optic nerve of one side. Pressure on the optic nerve leads to atrophy and cuts off communication with the vaginal space; the other vaginal space being open, papill- œdema occurs here.

Paton has recently published statistics of 252 cases of cerebral tumour with optic neuritis derived from the clinical records of the

Analysis of 252 Cases of Cerebral Tumour (Paton).

Site	Number	Confirmed by operation or p.-m.	No neuritis	Percentage of cases showing no neuritis	Percentage amongst confirmed cases	Post-neuritic atrophy	Slight neuritis	Marked neuritis	Average swelling*	Highest swelling observed*	Neuritis equal in two eyes*	Neuritis greatest in homo-lateral eye*	Neuritis greatest in contra-lateral eye*	Neuritis developed first in homo-lateral eye†	Neuritis developed first in contra-lateral eye†
Frontal	32	20	4	12·5	10	6	1	21	4·27 D	8–9 D	15	2	3	6	4
Parietal	28	23	4	14·285	13·636	1	5	18	3·56 D	6 D	7	6	2	3	4
Temporo-sphenoidal and occipital	14	12	0	0	0	0	0	14	4·72 D	7 D	9	4	0	1	0
Subcortical	29	26	11	37·931	37·037	1	5	12	4·227 D	8 D	9	2	4	5	1
Optic thalamus and mid-brain	16	13	4	25	23·077	2	0	10	4·1 D	7 D	5	2	1	1	—
Cerebellum	41	24	0	0	0	13	1	27	4·416 D	7 D	16	5	2	4	2
Extra-cerebellar	13	12	2	15·377	8·3	0	0	11	5·033 D	7 D	6	-	3	2	1
Pons and medulla	23	14	10	43·478	35·714	3	1	9	5·25 D	6·5 D	5	1	1	1	1
Basal	6	3	3	50	100	1	0	2	—	—	—	—	—	—	—
Totals	202	147	38	18·81	18·05	27	13	124	4·392 D	—	72	23	16	23	13
Conditions simulating tumours	5	5	—	—	—	—	—	—	—	—	—	—	—	—	—
Diagnosed as tumour but site undetermined	45	0	2	—	—	10	2	31	4 D	8 D	11	—	—	—	—
Totals	252	152	40	—	—	37	15	155	4·347 D	—	83	—	—	—	—

* The numbers given in these columns are calculated from those cases only in which definite and accurate measurements are available.

† The number of cases in which it is possible to make these observations is for obvious reasons very limited.

National Hospital, Queen Square, from 1899 to 1905. The analysis of these cases is shown in the table. In 202 of the cases the localisation is definite; in 148 it was confirmed by operation or autopsy. Every case of temporo-sphenoidal tumour showed optic neuritis. All but 2 out of 54 cerebellar cases showed neuritis, and these 2 were extra-cerebellar tumours. Cases of frontal and parietal tumour show nearly the same percentage without neuritis—frontal 12·5 per cent., parietal 14·285 per cent.; if cases with very slight neuritis are added, the percentages become respectively 15·625 and 28·57. In subcortical tumours a very large number (37·931 per cent.) show no optic neuritis, and this is even more marked in tumours of the pons (43·48 per cent). In these cases the development of optic neuritis coincides with invasion of the cortex or of the lateral ventricle. Paton considers that the relative infrequency of papillœdema in subcortical and pontine tumours is due to the low metabolic demand and low vascularity of the conducting tissues of which these structures are chiefly composed. An analysis of the nature of the growths leads to the conclusion that it is of little importance except in so far as the nature of the tumour determines its position. Paton finds that neither the side of greater swelling of the disc nor of earlier development of neuritis are of definite diagnostic importance as to the side of the tumour. There is no reason to suppose that myopia has any deterrent effect upon the development of papillœdema as has been thought.

Gowers.—The Ophthalmoscope in Medicine, London, 1904. Oppenheim.—In Nothnagel, ix, 3, 1891. Bruns.—Geschwülste des Nervensystems, Berlin, 1897. Edmunds and Lawford.—T. O. S., iv, 1884. Grösz.—Wiener Med. Presse, 1897. Oppenheim.—A. f. Psych., xxi, xxii. Williamson.—Brain, xix, 1896. *Kampherstein.—K. M. f. A., xliii, 1905. Hughlings Jackson.—R. L. O. H. Rep., vii, 1873. Field.—Brain, iv, 1881. Ulrich.—A. f. A., xxii, 1891. Rey.—Monatsschrift f. Psych., xv, 1904. Nonne.—Deutsche Z. f. Nervenheilk. xvii, 1904. Paton.—T. O. S., xxviii, 1908.

Syphilis.—Kampherstein in 200 cases of choked disc found 23 (12 per cent.) dependent upon syphilis—8 gummatous meningitis, 15 other syphilitic cerebral affections. In no case was it probable that disease of the cerebral arteries was the cause; in one case it was unilateral. Schott found syphilis the cause in 17·7 per cent. (50 cases). Uhthoff in 100 cases found syphilis the cause in 14—8 basal gummatous meningitis, 4 gummatous tumours; in 150 autopsies from the literature lues cerebri was the cause in 15 (10 per cent.). Uhthoff found unilateral papillœdema in one case, and a recurrence in one case. Hulke records a unilateral case with a gummatous tumour in the region of the sella turcica. The prognosis is unusually favourable, and complete restoration, both functional and ophthalmoscopic, may occur (Uhthoff, Nonne, Hutchinson, Oppenheim). Papillœdema is commonest in gummatous meningitis of the base, rarer when the convexity is affected (Gowers, Nonne). It may be the sole sign of cerebral syphilis (Förster).

Kampherstein.—K. M. f. A., xliii, 1905. Uhthoff.—A. f. O., xl, 1, 1894. Nonne.—Syphilis u. Nervensystem, Berlin, 1901.

Tuberculosis.—In 200 cases Kampherstein found tuberculosis 9 times—8 solitary tubercle, 1 chronic tuberculous meningitis. In

one case there was tubercle of the iris. Tubercle of the choroid is commoner, but less common than in general tuberculosis. Garlick found it once in 26 cases, Heinzel never in 41 cases. Kabsch found tubercle in the optic nerve sheath (*cf.* v. Michel, Vol. II, p. 683).

The commonest site of solitary tubercle is the cerebellum or pons. Kampherstein collected 32 cases—cerebellum 13, frontal 5, temporal 2, optic tract 1, cerebral peduncle 2, colliculi 3, thalamus 1, lenticular nucleus 1, paracentral lobule 2, centrum ovale 1, third nucleus 1. Papillœdema was present in 19 cases, 11 of these being affections of the cerebellum, 3 of the frontal lobes, 2 of the thalamus, 1 of the optic tract. The relative frequency of tubercle of the cerebellum is shown by Kraus—in 100 cases, tubercle 22, glioma 18, abscess 10, cysts 7, unknown tumours 13, endothelioma, gumma, blood-clot, softening, etc. 30; in 78 cases papillœdema was present in 66.

KAMPHERSTEIN.—K. M. f. A., xliii, 1905. GARLICK.—Med.-Chir. Trans., 1879. HEINZEL.—Jahrb. f. Kinderheilk., 1875. KABSCH.—Dissertation, Würzburg, 1891. SCHOELER. —K. M. f. A., xi, 1873. KRAUS.—Med. Jl., 1895.

Brain abscess.—In 200 cases Kampherstein found brain abscess in 7 (3½ per cent.), mostly due to middle-ear disease. Knapp found abscess of the temporal lobe commonest—66 per cent. I have seen a case of bilateral papillœdema in right orbital cellulitis with abscess of the right temporal lobe. The comparative rarity of choked disc in brain abscess is attributed by Deutschmann to the thick capsule which prevents the distribution of phlogogenic material. Gowers considers the probability dependent upon the rapidity of onset. Kampherstein has collected 155 cases of brain abscess, and has found optic neuritis in 37, hyperæmia in 4, atrophy in 1, retinal hæmorrhage in 1, papillœdema in 42, no ophthalmoscopic note in 70. Papillœdema occurs therefore in 25—30 per cent. of all cases. Cases of recurrence of papillœdema after operation, due to proliferation of granulation tissue, are reported by Gussenbauer, Jackson, Benedickt, Pflüger, Hadden, Norton, Murray, Gowers, Knapp, and others. Unilateral papillitis (6 times) and papillœdema (twice) occur occasionally (Lohmeyer, Saenger and Wiesinger).

KAMPHERSTEIN.—K. M. f. A., xliii, 1905. KNAPP.—Z. f. Ohrenheilk., xxvi. DEUTSCH-MANN.—Ueber die Neuritis optica, Jena, 1887. GUSSENBAUER.—Prager med. Woch., 1885. LOHMEYER.—Berliner klin. Woch., 1890. SAENGER AND WIESINGER.—Münchener med. Woch., 1893. KÖLPIN.—Deutsche Z. f. Nervenheilk., xxv, 1905. SAENGER.—A. f. Psych., xli, 1905.

Cysticercus.—Kampherstein found 2 cases of cerebral cysticercus in 200 of papillœdema; they have been reported by Jacoby, who has collected with ophthalmoscopic details 24 cases from the literature. In 13 cases the parasite was in the fourth ventricle and there was internal

hydrocephalus; in 6 of these the fundus was normal (*cf.* Cramer), 7 had neuritis. In 4 cases the cysticercus was in the brain substance; in 3 the fundus was normal. In 2 it was in the lateral ventricle, one having normal, the other abnormal fundus.

JACOBY.—K. M. f. A., xli, 1903. CRAMER.—Versamml. der Irrenärzte, Hannover, 1897. HEBOLD.—A. f. Psych., xv. HARTMANN.—Wiener klin. Woch., 1902. JACOBI.—B. d. o. G., 1903. SATO.—Deutsche Z. f. Nervenheilk., 1904. WOLLENBERG.—A. f. Psych., xl, 1905. v. KRÜDENER.—St. Petersb. med. Woch., 1905. SCHLAGINTWEIT.—Deutsche Z. f. Chir., lxxvi, 1905. HENNEBERG.—Charité Annalen, xxx, 1906; Neurol. Centralbl., 1906.

Aneurysm of cerebral arteries.—Papilloedema is rare in cases of cerebral aneurysm, and only post-mortem examination suffices to establish the diagnosis (Beadles). In Holmes's case there was a growth of the pituitary body as well as aneurysm of the internal carotid. v. Michel's case was not an aneurysm. There was papilloedema in the cases of large aneurysms reported by Anderson and Bramwell. Waldo's case, an aneurysm of the size of a cherry on the right internal carotid, had bilateral papilloedema. Hutchinson, in a case of aneurysm of the basilar artery, found hæmorrhages of the retina. Hæmorrhage into the optic nerve sheath is recorded by Bramwell, Mott and Stedman, Hale White.

ANDERSON.—Lancet, 1885. BRAMWELL.—Edin. Med. Jl., 1886; Intra-cranial Tumours, Edinburgh, 1888. BRUCE AND DRUMMOND.—Rev. of Neurol., ii, 1904. HOLMES.—Am. Jl. of Med. Sc., 1864. HUTCHINSON, JR.—T. O. S., ix, 1889. JEAFFRESON.—Lancet, 1879. v. MICHEL.—A. f. O., xxiii, 2, 1877. MOTT AND STEDMAN.—Lancet, 1889. ROSS.—Canada Med. and Surg. Jl., 1883. WALDO.—Brit. Med. Jl., 1903. HALE WHITE.—Clin. Soc. Trans., 1895. A. FUCHS.—Neurol. Centralbl., 1904. SILCOCK.—T. O. S., xxiv, 1904. *BEADLES.—Brain, xxx, 1907.

Intra-cranial hæmorrhage.—Papilloedema is probably much commoner from this cause than would be gathered from the reported cases, since an ophthalmoscopic examination is comparatively rarely made. There are two groups of cases, those due to injury and those due to disease of the vessels, hæmorrhagic pachymeningitis, purpura, etc. Cases of fracture of the skull with choked discs have been recorded fairly often (Liebrecht, Gowers, Panas, Pflüger, Talko, Priestley Smith, Silcock, Remak, and others). In 7 cases which were examined post mortem there was hæmorrhage in the subdural space and in the vaginal space of the optic nerve. Panas observed papilloedema 5 days, Pflüger 10 days after the injury.

Remak found 3 cases of hæmorrhage from ruptured aneurysm in the neighbourhood of the Sylvian fissure (Mackenzie, Samt, Fürstner). Other cases of neuritis associated with spontaneous intra-cranial hæmorrhage are recorded by Gowers, Hughlings Jackson, Bristowe, Schiess-Gemuseus, v. Michel, Manz, Liebrecht, Hermann.

LIEBRECHT.—Münchener med. Woch., 1903. REMAK.—Berliner klin. Woch, 1886. PRIESTLEY SMITH, SILCOCK.—T. O. S., iv, 1884. HERMANN.—C. f. A., xvii, 1893. v. HASELBERG.—Charité Annalen, xxvii, 1904. BACHAUER.—Deutsche med. Woch., 1904. CHAILLOUS.—Rec. d'O., 1905. BUNGE.—Münchener med. Woch., 1905. FERENCZI.— Neurol. Centralbl., 1905.

Pathological anatomy.—The earliest stage of papilloedema rarely becomes available for microscopic examination. In dealing with the

theories of the mode of production of choked disc it will be seen that there has been much dispute as to whether the condition is primarily an œdema or an inflammation. The general consensus of opinion is now in favour of the former view. According to Knies there is at first only œdema of the nerve head with no changes in the extra-ocular portion of the nerve. Most observers of the cases usually examined, viz. those of a later stage, find definite evidences of inflammation involving the nerve, the lamina cribrosa, and the nerve sheaths. Elschnig has examined a very large number of cases, and has invariably found changes in the nerve and its sheaths similar to those of papillitis proper, the only difference being the pronounced œdema of the non-medullated portion of papilla. The lamina cribrosa is almost always bowed forwards as though by pressure from behind, and this is the
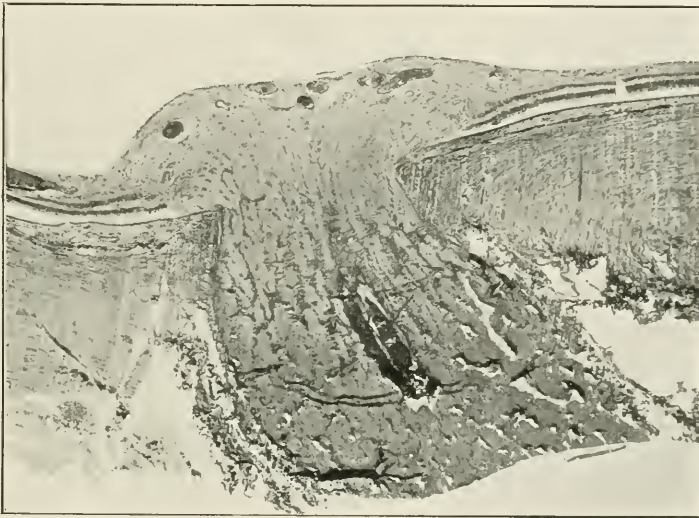
Fig. 847.—Papillœdema.
From a photograph by Coats.

most constant anatomical difference between papillitis and papillœdema (Elschnig).

Sourdille found only slight and scattered infiltration of the sheaths with round cells. There was also slight infiltration of the septa, most marked near the lamina cribrosa and diminishing posteriorly. The glia cells showed proliferative changes with many mitotic figures and astrocytes. Rochon-Duvigneaud, too, found but sparse infiltration, though œdema was conspicuous; there was the same neuroglial proliferation. Ginsberg examined six cases with similar results—slight infiltration, but always marked glial proliferation. The latter is sometimes limited to the periphery of the orbital part of the nerve (Sourdille, Ginsberg), sometimes spread over the whole diameter. The convexity forwards of the lamina was absent in two of Ginsberg's cases.

The medullary sheaths of the fibres show degenerative changes or complete atrophy. The peripheral bundles are most affected, often most near the globe (Rochon-Duvigneaud) though not always (Elschnig). There is little extension of the degenerative process beyond the limits of the most affected areas (Rochon-Duvigneaud, Sourdille, Ginsberg), and sometimes isolated areas entirely escape (Sourdille, Elschnig). Elschnig describes thickening of the choroidal portion of the lamina cribrosa, but this was absent in Ginsberg's cases.

Kampherstein has recently examined 51 eyes collected by Uhthoff. In 65 per cent. there was distension of the vaginal space; Elschnig in 26 cases found about the same proportion. Only 35—40 per cent. showed extreme dilatation, the remainder being little beyond the physiological limits of variation. It is noteworthy that the earliest cases showed on the whole least distension. The subarachnoid space was most affected, the arachnoid being pressed against the dura and in later stages fused with it. Generally the dilatation was most marked near the globe, but there were also cases in which the site of entry of the central vessels was most involved.

In 38 out of 51 cases there were inflammatory signs in the sheath spaces, absent, however, in the earliest stages examined; they consist generally of irregularly distributed patches of round cells. Occasionally the whole space was infiltrated, sometimes uniformly, generally most in the central part, the arachnoid being much thickened. In 2 cases the physiological ampulla was filled with granulation tissue, whilst the rest of the vaginal space was free from inflammation. Papilloedema which has gone on to atrophy is always accompanied by inflammatory signs in the sheath spaces.

Inflammatory changes were found in the nerve in 28 cases (56 per cent.), varying enormously in intensity. In many cases they were very slight and appeared to vary independently of the changes in the sheaths. In the least marked cases there were aggregations of lymphocytes in the septa and their finer expansions. The nuclei of the individual nerve bundles are increased by reactive proliferation of the glia cells, always densest just behind the lamina cribrosa.

The chief feature is oedema of the nerve (Kampherstein), more or less definite in 60 per cent. of cases. Subpial oedema (Ulrich) was seldom found. The most frequent and most characteristic form was interfascicular oedema. The broadest spaces, traversed by fine fibrils, were found at the nodal points of the septa, where several meet; they often contain granular masses and myelin (Schmidt-Rimpler). There was also oedema within the septal tissue, and it was specially notable around the central vessels. Intra-fascicular oedema is much rarer, and always associated with the interfascicular form. Oedema is present even in the atrophic stage. In 19 cases oedema and inflammatory signs were present side by side; according to Liebrecht the more marked the inflammatory infiltration the less are the lymph spaces distended. Kampherstein paid special attention to artefacts, which, as is well known, are very liable to be regarded as pathological manifestations in the optic nerve (v. Vol. II, p. 657).

The atrophy of papilloedema is a neuritic degeneration. The septa are thickened, the nerve-fibres disappear slowly, and neuroglial proliferation is brought prominently into view by the increased nuclear richness of the tissues. The loss of nerve-fibres causes shrinking. The vessels are normal in the early stages: in the later stages the walls become thickened, and the smaller vessels and capillaries are increased in number.

Kampherstein was able to examine the lamina cribrosa in 42 cases; in 33 it was bowed forwards, but a distinction must be made between early and late cases, for in the atrophic stage the lamina returns to almost its normal situation (4 cases). It is always convex anteriorly in fresh cases (88 per cent.; Elschnig—41 out of 44 cases; Liebrecht—in all of 12 cases). This feature was first emphasised by Schweigger. It is the choroidal portion of the lamina which is most affected, the scleral portion being denser and showing the condition only in a modified manner. Enormous bowing forwards of the lamina is shown in a case reported by Yamaguchi. Though the normal situation is recovered in the atrophic stage yet there seems to be an excess of tissue, the fibres showing convolutions and irregularities.

In 15 out of 42 cases there was no sign of inflammation in the papilla; there is simply intense oedema. Round and oval spaces are found between the fibres, partly filled with coagulated exudate. The physiolological cup is smaller and shallower or quite obliterated. In the cases with inflammatory signs these consisted of aggregations of round cells in the perivascular spaces, and to a less degree on the fibrous processes derived from the lamina cribrosa. The capillaries are increased and their walls thickened.

New-formed fibrous tissue first makes its appearance in the physiological cup as shown by increase of nuclei around the vessels. Fibroblasts, spindle shaped with long oval nuclei, stretch forwards, so that a thin membrane is formed over the surface of the swelling.

Occasionally the shape of the papilla is unusual, e. g. only one side is swollen (Kampherstein).

The oedema extends a considerable distance into the retina, often a papilla diameter or more. The nerve-fibre layer is most involved but the other layers are almost always affected, especially the internuclear and inner nuclear layers. There may be a fold at the edge of the disc so that there is a small detachment of the retina from the choroid.

Kampherstein's chief results may be tabulated as follows:

Vaginal space not distended			.	.	.	19
,,	,,	distended	.	.	.	32
,,	,,	much distended	.	.	.	19
,,	,,	showed infiltration	.	.	.	38
,,	,,	,,	,,	and distension	.	23
,,	,,	,,	,,	without distension		15
Optic nerve apparently normal			.	.	.	5
,,	showed oedema .		.	.	.	30
,,	,,	infiltration	.	.	.	28
,,	,,	oedema and infiltration		.		19

out of 51 cases

Lamina cribrosa bowed forwards . . . 33
,, choroideæ ,, . . . 1
,, cribrosa not bowed forwards . . 9
Papilla showed infiltration 27
,, ,, no infiltration . . . 15
out of 42 cases.

Pathogenesis.—Türk (1853) first drew attention to ophthalmoscopic changes in a case of brain tumour, viz. retinal hæmorrhages. It was v. Graefe (1860), however, who first described choked disc and its relationship to intra-cranial disease. He held that tumour causes pressure either directly or indirectly upon the cavernous sinus, thus producing congestion of the ophthalmic vein and central vein of the retina, leading to œdema of the papilla and hæmorrhages. Doubt was thrown upon this explanation by Sesemann, who emphasised the communications between the ophthalmic vein and the veins of the face. Gurwitsch and Judeich still adhered to the view that the orbital venous blood, and indeed that of the forehead and cheeks, normally returned by way of the cavernous sinus. Judeich, however, pointed out that the sinus is protected from compression by its dense covering.

Schmidt-Rimpler advanced another mechanical theory, based upon Schwalbe's demonstration of the continuity of the subarachnoid space with the vaginal space of the optic nerve. According to him increased intra-cranial pressure impedes the flow of lymph, so that fluid is pressed out of the distended vaginal space into the lamina cribrosa. Manz doubted the inflow of fluid into the lamina and considered that the vessels only were compressed by the distension of the sheath. Deyl explained papillœdema by kinking of the central vein and its compression due to the hydrops vaginæ nervi optici.

Parinaud and Ulrich adopted a similar mechanical theory without having recourse to distension of the sheath. The obstruction to lymph flow towards the cranium causes œdema of the optic nerve which eventually produces papillœdema.

The anatomical observations failed to give unanimous support to any of these mechanical theories, and gave rise to others founded upon an inflammatory cause. Leber (1881) advanced the view that the intra-cranial disease led to the development of phlogogenous material which passed into the cerebro-spinal fluid and so to the nerve sheath, nerve and papilla. Inflammation was set up, with œdema and infiltration as secondary manifestations. Deutschmann has endeavoured to confirm this theory by experiment, and it has been supported on anatomical grounds by Edmunds and Lawford, Gowers, Zellweger and Haab, Scimeni, Elschnig, and others. Baas attempts to combine both mechanical and inflammatory theories for the explanation of what he calls " Stauungspapillitis."

Vasomotor theories have been advanced or supported by Benedickt, Hughlings Jackson, and others. Benedickt considers that papillœdema is a vasomotor disturbance brought about by pathological changes in the sympathetic. Loring and Hughlings Jackson consider that nerve centres which control the circulation and nutrition of the

optic nerve are compressed; Adamkiewicz, on similar grounds, regards the condition as a neuroparalytic inflammation.

Rochon-Duvigneaud regards the condition as the result of long-continued action of stagnant lymph. Krückmann also considers that it is a primary œdema; he thinks that the products of metabolism contain positive chemotactic substances which lead to leucocytosis. The stagnant products also poison the tissues and cause their disintegration.

Experimental investigation has been carried out by Manz, Parinaud, v. Schultén, Deutschmann, Merz, Kampherstein, and others. v. Schultén found that when the intra-cranial pressure reached 40—60 mm. Hg. in rabbits the physiological cup became shallower, the arteries were narrowed and the veins dilated. With pressures of 100—120 mm. Hg. the changes were increased. He considered that fluid passed into the vaginal space and compressed the nerve and central vein. Merz experimented on rabbits and dogs. The intra-cranial pressure was raised, as in v. Schultén's experiments, by fluid. With slight increase (15—20 mm. Hg. in dogs, 10—15 mm. Hg. in rabbits) arterial anæmia and venous hyperæmia were produced. In 2—3 hours the edges of the disc became blurred; in 8—12 hours there was much swelling and the other features were exaggerated. Kampherstein and Heine repeated Merz's experiments, but failed entirely to confirm them; only if the experiment was continued after the death of the animal was definite protrusion of the papilla noticeable.

Amidst the conflicting facts and theories of papillœdema the fundamental importance of increased intra-cranial pressure stands out prominently. The clinical evidence derived from cerebral surgery has of recent years thrown this factor into brilliant relief. All those who have had opportunities of watching the extraordinary effect of the relief of intra-cranial pressure upon a choked disc must agree that no theory which leaves this element out of account requires any further consideration. Comparatively little has been published on the changes in the fundus following the relief of pressure, but attention may be directed to the papers of Taylor and Paton. It is particularly striking that merely opening the skull fails to produce any effect upon the disc unless pressure is relieved. Thus, it is usual to perform the operation in two stages; no change occurs until after the second stage—that of opening the dura mater—has been completed.

Apart from these clinical details little light has been thrown on this obscure problem. It must be admitted that it is still unsolved. It is possible that intra-cranial pressure acts in a manner entirely different from that suggested in the theories as yet advanced, possibly in some physico-chemical manner. Thus, Dean has shown that the brain substance compressed by a glass disc introduced into the skull contained 3 per cent. more water than normal. It is a well-known fact that the brain surrounding a tumour is extremely œdematous. A reasonable explanation has been brought forward by Cannon. Loeb has shown that if one leg of a frog is ligatured so that it is deprived of its blood supply the muscles take up water, so that the gastrocnemius in 18 hours contains 1—3 per cent., in 48 hours 15 per cent., and in

7 days 25—40 per cent. more water than the other. The assumption of water by a muscle deprived of its blood supply is due to chemical changes in the muscle increasing the internal osmotic pressure, and these chemical changes are probably due to lack of oxygen. The normal osmotic pressure of frog's gastrocnemius is equal to that of isotonic saline solution, *i.e.* about 5 atmospheres. If the muscle is placed in 4·9 per cent. NaCl solution, which has an osmotic pressure of over 30 atmospheres, it first loses water, as might be expected, but later absorbs it against this enormous pressure. Cannon has shown that brain substance acts in the same manner. He concludes that "under circumstances of non-nutrition the brain will take up water from a solution isotonic with blood, and will thereby exert a pressure sufficient to exclude the blood from the cerebral vessels." The pressure of a foreign body, as Hill has emphasised, squeezes the blood out of a given volume of brain substance, whilst the neighbouring areas are also badly supplied. These areas will absorb water owing to the retention of chemical products which alter their osmotic pressures. They will therefore swell and thus press on other areas, reducing them to the same condition, so that a circulus vitiosus is set up. Diminution of the intra-cranial space by one twelfth eventuates in death, but doubtless before this occurs protective mechanisms are brought into play. Asphyxiation of the vasomotor centre is probably one whereby the blood-pressure is raised, the vascular channels are re-opened, and the deleterious products are swept away. Nevertheless it is clear that under pathological conditions a purely local compression may be transformed into a widely spread one. In this way it is by no means improbable that the blood flow in the basal arteries and sinuses is impeded.

In those animals in which the ocular blood supply is mainly of cerebral origin one may expect high intra-cranial pressure to be accompanied by slowing of the orbital and intra-ocular blood flow, with resultant malnutrition. Why the œdema of the optic nerve should manifest itself almost entirely at the intra-ocular end must be due to local causes, and these are not far to seek. A very slight œdema of the nerve at the lamina cribrosa will compress the capillaries, lead to greater anæmia and greater œdema, until finally the larger vessels are also compressed. The fact that papillœdema is at first a pure œdema is in favour of this view. It is only later, when the products of disintegration have accumulated and acted as a chemical stimulus, that infiltration with leucocytes occurs. It would be unwise to press the hypothesis too far, but it may be of value in guiding future researches.

Türk.—Z. d. Gessellsch. d. Wiener Aerzte, ix, 1853. v. Graefe.—A. f. O., vii, 2, 1860; xii, 2, 1866. Hughlings Jackson.—R. L. O. H. Rep., iv, 1863. Manz.—K. M. f. A., iii, 1865; A. f. O., xvi, 1870; Deutsches A. f. klin. Med., ix, 1871; B. d. o. G., 1874. Benedickt.—Allgem. Weiner med. Zeitschrift, 1868. Schmidt-Rimpler.—A. f. O., xv, 2, 1869; A. f. A., xviii, 1888. Sesemann.—A. f. Anat. u. Physik., 1869. Lorenz.—Amer. Jl. of Med. Sc., 1873. Herzog.—K. M. f. A., xiii, 1875. Leber.—In G.-S., v, 1877. Kuhnt.—B. d. o. G., 1879; Internat. Congress, London, 1881. Parinaud.—Ann. d'Oc., lxxxii, 1879. Treitel.—A. f. O., xxv, 1880. Loring.—New York Med. Jl., 1882. Lawford.—T. O. S., iii, 1883; iv, 1884; vii, 1887. v. Schultén.—A. f. klin. Chir., xxxii, 1885. Deutschmann.—Ueber Neuritis optica, Jena, 1887. Zellweger.—Dissertation, Zürich, 1887. Ulrich.—A. f. A., xvii, 1887; xviii, 1888; xxii, 1890. Dean.—Jl. of Path., i, 1893. Adamkiewicz.—

Neurol. Centralbl., 1893; 1905; Z. f. klin. Med., xxviii, 1895. GREEFF.—In Orth's Lehrbuch, 1903. ROCHON-DUVIGNEAUD.—A. d'O., xxv, 1895. *ELSCHNIG.—A. f. O., xli, 2, 1895; Wiener klin. Rundschau, 1902. HOCHE.—A. f. A., xxxv, 1897. KRÜCKMANN.—A. f. O., xlv, 3, 1898. LOEB.—Pflüger's Archiv, lxxi, 1898. DEYL.—Wiener klin. Rundschau, 1899. BAAS.—Z. f. A., ii, 1899. JUDEICH.—Z. f. A., iii, 1900. MERZ.—A. f. A., xli, 1900. SOURDILLE. —A. d'O., xxi, 1901. CANNON.—Amer. Jl. of Physiol., vi, 1901. ELSCHNIG.—Wien klin. Rundschau, 1902. LIEBRECHT.—B. d. o. G., 1902; Münch. med. Woch., 1903; Neurol. Centralbl., 1904; K. M. f. A., xlii, 1904. GINSBERG.—Grundriss d. Path. Hist. d. Auges, Berlin, 1903. TAYLOR.—T. O. S., xiv, 1894. YAMAGUCHI.—K. M. f. A., xli, 1903. VAN GEUNS.—A. f. A., xlviii, 1903. FLATAU.—A. f. klin. Med., lxxvii, 1903. FLEMMING.—Rev. of Neurol., 1904. UHTHOFF.—Neurol. Centralbl., 1904. *KAMPHERSTEIN.—K. M. f. A., xlii, 1904; xliii, 1905. REICHARDT.—Deutsche Z. f. Nervenheilk., xxxiii, 1905. OPPENHEIM. —Monatschrift. f. Psych., xviii, 1905. FÜRSTNER.—A. f. Psych., xl, 1905. PATON.—T. O. S., xxv, 1905. SAENGER.—Münch. med. Woch., 1905; K. M. f. A., xlv, 1907. PARSONS.—The Ocular Circulation, London, 1903. MORAX.—Ann. d'Oc., cxxxviii, 1907. LEVINSOHN.— A. f. O., lxiv, 1906. v. KRÜDENER.—A. f. O., lxv, 1906.

Amaurotic family idiocy.—The first case of this disease was shown at the Ophthalmological Society of the United Kingdom in 1881 by Waren Tay. It was exhaustively investigated clinically from the neurological side by Sachs. All the published cases were collected and collated by Falkenheim in 1901. The pathological anatomy of the eyes has been investigated by Treacher Collins, Shumway and Buchanan, and myself; that of the nervous system by Sachs, Risien Russell, Hirsch, Peterson, Frey, Schaffer, Spiller, Spielmeyer, and most exhaustively by Gordon Holmes. The chemistry of the central nervous system in the disease has been investigated by Mott.

The disease occurs exclusively in children of Jewish birth. The first symptoms appear at about 3—6 months, the child showing weakness in the muscles of the neck and back. Simultaneously or at a short interval defective sight is noticed. Muscular weakness and failure of vision increase and marasmus causes death usually within 2 years. The ophthalmoscopic appearances resemble those of embolism of the central artery of the retina. They are symmetrical and are almost identical in all cases. There is a large white area at the posterior pole with a maroon-coloured spot in the centre corresponding with the macula, rather darker in colour than the cherry-red spot of embolism. In the early stage the discs may be normal. Later all stages of optic atrophy are passed through up to complete blindness with chalky white discs and small vessels.

The brain is peculiarly firm and of the consistence of gutta-percha. Microscopic examination shows extraordinarily widespread degeneration of the cells with relatively less degeneration of the fibres than might be expected. Mott agrees with Gordon Holmes that the essential histological feature is a progressive loss of the Nissl substance of all the neurons of the body. Histo-chemical observations support the view that this substance is a nucleo-proteid. Mott examined 3 brains from cases of amaurotic family idiocy. All showed decrease of nucleo-proteid, which may be associated with the disappearance of the Nissl substance in the neurons, and increase of simple proteids, which may be correlated with the increase of glia fibrils, which give the unusual firmness of consistency. There is little doubt that the disease primarily affects the nerve cells and particularly the interfibrillar protoplasm (Gordon Holmes). It is not due to arrested development, and the

negative results of bacteriological examination suggest that it is not
due to bacterial toxins, a view which is further supported by its family
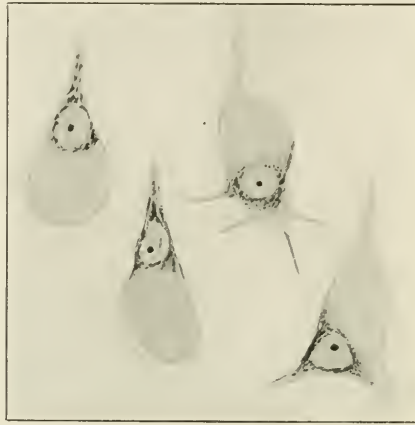
Fig. 848.—Amaurotic Family Idiocy.

Poynton, Parsons, and Gordon Holmes, Brain, 1906. Pyramidal cells from
the precentral gyrus stained by Nissl's method. The cell bodies are swollen and
the nuclei are excentric. The tigroid has disappeared, and the only stainable
substance in the cells is granular *débris* around the nucleus.

nature, its occurrence only in the Jewish race, and its constant appear-
ance within a certain short period of life. The disease is probably due
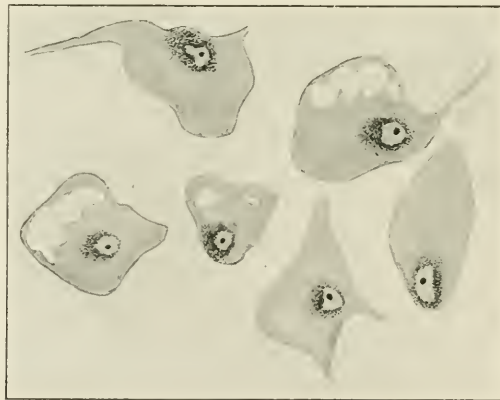
Fig. 849.—Amaurotic Family Idiocy.

Poynton, Parsons, and Gordon Holmes, Brain, 1906. Motor cells of the
ventral horn of the spinal cord stained by Nissl's method. The cells are very
much swollen, their nuclei are excentric, the tigroid has disappeared, and the
protoplasm is vacuolated.

to some inherent bio-chemical property of the protoplasm of the cells.
The cell changes have not the characters of a simple atrophy; in fact

they seem to be due to an excessive growth of the protoplasm which later undergoes degenerative changes.   This fact is not in favour of

FIG. 850.—AMAUROTIC FAMILY IDIOCY.

Poynton, Parsons, and Gordon Holmes, Brain, 1906.   Two Betz cells and a pyramidal cell, the central one of the precentral gyrus stained by Bielschowsky's method.   Note the partial disintegration of the neurofibrils in the region of the nucleus and the hour-glass shape of the pyramidal cell.

FIG. 851.—AMAUROTIC FAMILY IDIOCY.

Poynton, Parsons, and Gordon Holmes, Brain, 1906.   Motor cells of the ventral horn of the spinal cord stained by Bielschowsky's method.   Vacuolation of the protoplasm of the cells and partial breaking-up of the neurofibrils are visible.

Sachs's hypothesis that the pathology of the disease can be described by the term "abiotrophy," a term suggested by Gowers to represent an

inherent defective vitality of the cell, nor of Schaffer's suggestion that
it may be explained by Edinger's "Ersatztheorie," which assumes

Fig. 852.—Amaurotic Family Idiocy.
Poynton, Parsons, and Gordon Holmes, Brain, 1906. A cross section of the
upper cervical region of the spinal cord stained by the Weigert-Pal method.
Partial degeneration of the crossed pyramidal tracts is visible, but the spino-
cerebellar tracts and the dorsal columns appear normal.

that elements which are inherently feeble undergo degeneration when
exposed to the strain of life to which they are not normally resistant.

Fig. 853.—Amaurotic Family Idiocy.
Poynton, Parsons, and Gordon Holmes, Brain, 1906. A portion of the lateral
column of an upper cervical segment stained by Weigert's neurolgia method. It
shows sclerosis limited to the area of the crossed pyramidal tract, and the absence
of neuroglial proliferation in the region of the direct cerebellar tract.

I have examined the eyes of two cases. A minute hole could be
demonstrated macroscopically in one eye of Case 1. On examination

of the other sections of the eye of this case, which was not opened until after hardening and freezing, no hole could be seen. Even in the sections of Case 3, which had been submitted to much more drastic
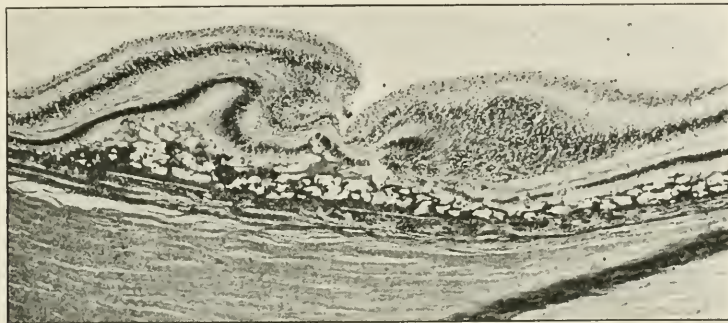
FIG. 854.—AMAUROTIC FAMILY IDIOCY.

Poynton, Parsons, and Gordon Holmes, Brain, 1906. The macular region from Case 1. Note the folding of the retina: serial sections gave no evidence of an actual hole at the fovea centralis. The nerve-fibre layer is absent; the nuclear layers retain their normal disposition; the reticular layers are œdematous, only the neuroglial elements remaining. The retinal pigment epithelium has become desquamated, a result undoubtedly due to post-mortem change.

treatment, whilst the fovea was extremely thin, some tissue or exudate still filled in the fovea and held the parts together.

Microscopical examination shows that, apart from the retina and optic nerve, the eyes were normal.

FIG. 855.—AMAUROTIC FAMILY IDIOCY.

Poynton, Parsons, and Gordon Holmes, Brain, 1906. The macula from one eye of Case 3. Only the backs of the eyes were obtained, the retina being detached and folded. There is a definite hole at the fovea, which may be, and probably is, due to the dislocation of the parts subsequent to death. In other respects the features are the same as in the previous figure.

The retina in both cases shows degeneration of the ganglion cells and nerve-fibre layer over the whole area. At a distance from the macula there are far fewer ganglion cells than normal, so that there

can be little doubt that large numbers have completely atrophied. The nerve-fibre layer is extremely attenuated everywhere.

The minute histology is complicated by the presence of post-mortem changes. It has been shown by Birch-Hirschfeld and others that post-mortem changes are evident in the cells of the retina within a very few hours after death. In these specimens they are shown in the fusion and loss of detail of the rods and cones, but still more definitely in the separation in places of the retinal pigment epithelium from the underlying lamina vitrea. In one or two places there are minute folds of the retina, which is here separated from the choroid by an albuminous coagulum, in which loosened pigment cells are seen. These are
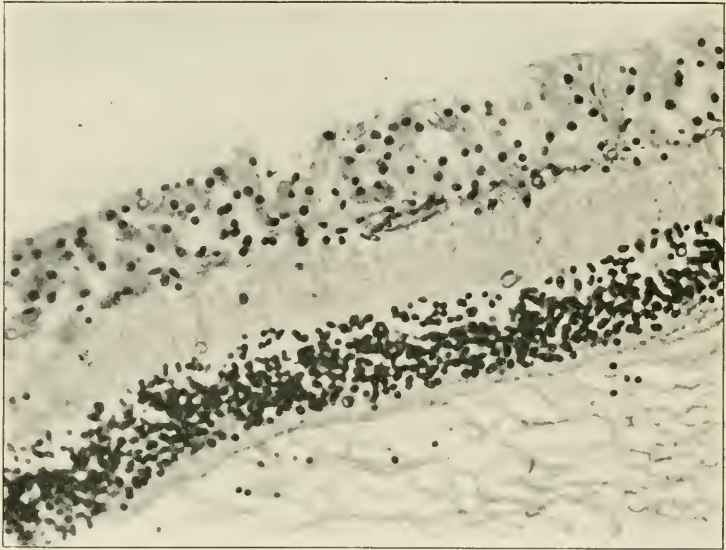
Fig. 856.—Amaurotic Family Idiocy.

Poynton, Parsons, and Gordon Holmes, Brain, 1906. Ganglion cells from the macular region in Case 3. Note that the cell bodies are swollen, and the nuclei are often eccentric. The nerve-fibre layer is absent: the reticular layers are seen to consist of spaced-out neuroglial elements.

undoubtedly post-mortem artefacts. These changes render difficult of accurate appreciation the changes found in the macular region.

Near the fovea in Case 1 the retina is much folded. That some of this folding is adventitious is shown by the fact that the chief fold is directed horizontally towards the optic disc—a condition which is commonly met with in eyes treated in the usual manner with formol, etc. The folding is, however, more marked than usual, and it is probable that some of it is actually due to the pathological processes which have been going on.

Everywhere but at the macula the nuclear and reticular layers show little change from the normal condition; they are evenly distributed, and of normal thickness. At the macula, owing to the folding, they

are cut obliquely in places, so that they appear to spread out and become thicker. That there is some true thickening is shown by distinct spacing out of the nuclei and fibres, the intervening spaces being filled with coagulated exudate. It is impossible to be certain that this is not of post-mortem origin, but the probability is in favour of some œdema of the retina here. It is well known that the macular region is especially prone to œdema in a considerable variety of pathological conditions, e. g. embolism of the central artery of the retina, section of the optic nerve anterior to the point of entry of the central vessels, etc. I have described the mechanism of this œdema elsewhere (v. p. 1185). The space behind the folded retina, separating it from the choroid, is filled with coagulated albuminous exudate, and there are well-marked post-mortem changes in the retinal pigment epithelium in this situation.

The minute cytology of the macular region fails to demonstrate anything abnormal in the cells of the nuclear layers. The ganglion cells are numerous and form several layers, in accordance with the normal anatomy of the part. There is no evident diminution in the number of ganglion cells in this region, but the individual cells show profound pathological changes. There is evidence to show that the ganglion cells of the retina are even more delicate than those of other parts of the central nervous system. As regards post-mortem changes, they are probably more exposed to damage than the latter. Whatever may be the relative value of these two factors it has been found impossible to demonstrate the changes in the cells so satisfactorily as in the central nervous system, but on the whole they are similar in type and are probably identical. The cells are much swollen and rounded, having lost their normal angular contour. The nuclei are often eccentrically situated, sometimes being apposed to the margin of the cell. There are often vacuoles in the cytoplasm, and Nissl granules are conspicuous by their absence. The nuclei stain well with nuclear stains, and the cytoplasm shows diffuse staining with Nissl reagents. There is a tendency towards concentration of the Nissl stain around the nuclei, as in the cells of the nervous system elsewhere, but definite granules are made out rarely and with difficulty. In the most degenerated cells the cytoplasm has shrunken away from the surrounding tissues, so that a clear space is seen.

The optic nerve is quite atrophic in the cases examined, and it is probable that there are very few intact fibres remaining. That the atrophy is not absolute is shown by the pupil reaction.

The explanation of the ophthalmoscopic appearances in amaurotic family idiocy cannot be considered to be placed beyond dispute by the anatomical examination. It is probable that the changes in the retina are fundamentally the same as in the central nervous system, i. e. that the ganglion cells suffer first and in exactly the same manner as elsewhere. This change is rapidly followed by the development of the typical ophthalmoscopic picture. It is to be noted that in the early stages, though the ophthalmoscopic picture is typical, there may be no definite evidence of optic atrophy. I have seen such cases, and have heard of others. The pallor of the disc and other signs

of atrophy, however, quickly supervene, and are generally present when the case first comes under treatment. It is probable that the white area around the fovea is due to œdema and slight folding of the retina, the latter being the immediate result of the former. The dark red spot at the fovea is due to contrast, as in most cases of embolism of the central artery. It is, perhaps, darker than is usually seen in these cases, and there is probably some congestion of the choroid, as shown by the anatomical examination, but it is difficult to be certain of this point. It is not probable that mere post-mortem change can cause a hole at the fovea, and it is highly probable that in the later stages the degeneration of the tissues goes on to complete atrophy, so that a hole is eventually formed by pathological processes, but it is impossible to be dogmatic upon this point.

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Cerebral degeneration with macular changes.—There is a group of cases, the first of which was reported by Rayner Batten, in which pigmentary changes at the macula, pallor of the disc, and contraction of the retinal vessels is associated with more or less pronounced idiocy in children who are not of Hebrew parentage. Other examples have been recorded by F. E. Batten, Still and Donald Gunn, Hirschberg, Stephenson, Mayou, Gessner and Müllberger. In most cases there is a history of syphilis in the parents but no signs of congenital syphilis in the children; in some there is consanguinity in the parents (Hirschberg, Stephenson, Mayou). The ocular condition and the mental deterioration arise at about the same period of life—from 7 to 14 years of age. The macula generally shows a reddish-black spot surrounded by an area of coarsely-granular pigmentation.

BATTEN.—T. O. S., xvii, 1897; xviii, 1898. HIRSCHBERG.—C. f. A., xxviii, 1904. MAYOU.—T. O. S., xxiv, 1904. VOGT.—Monatsschrift f. Psych., xviii, 1905. SPIELMEYER.—A. f. Psych., xl, 1905; Neurol. Centralbl., 1905, 1906. STOCK—B. d. o. G., 1906. GESSNER, MÜLLBERGER.—Münchener med. Woch., 1906. NETTLESHIP.—T. O. S., xxviii, 1908.

DISEASES OF THE MENINGES.

Tubercular meningitis.—Uhthoff found the following ocular complications in a large series of cases of tubercular meningitis: Moderate

optic neuritis in 25 per cent., of which 5 per cent. were unilateral; typical choked disc in 5 per cent., in which in all cases there was solitary tubercle in the brain complicating the meningitis ; descending neuritis in 4 per cent.; marked venous congestion in 5 per cent.; tubercle of the choroid in 10 per cent., half of which were complicated with papillitis. Thus nearly half the cases (49 per cent.) had ocular complications. Collation of previous observers' results gives 31 per cent. with optic neuritis. Cases of postneuritic atrophy are naturally rare in the literature (Hinselwood, Ihrig, and others), and were probably complicated with solitary tubercle. The literature gives 10 per cent. with choked disc, the majority complicated with solitary tubercle (Leimbach, Stephen Mackenzie, Warnicke, Elschnig, and others), or hydrocephalus, or more rarely basal meningitis (Elschnig). Previously reported cases give 9 per cent. with descending neuritis (Hock, Deutschmann, Edmunds and Lawford, and others). The clinical features of these cases are ambiguous. Anatomically there is often miliary tuberculosis of the nerve sheaths (v. Michel, Deutschmann, Sturm, Elschnig, and others). More commonly there is hydrops with perineuritic infiltration, not definitely tubercular in character. In a considerable number of cases there was solitary tubercle of the optic nerve or basal tracts, as, for example, the chiasma with temporal hemianopia (Hjort, Bamberger and Lütkemüller, Muratow, v. Herff). Cargill and Mayou's case with solitary tubercle of the choroid associated with miliary tubercle of the meninges is unique. Venous hyperæmia of the retina without signs of papillitis has been more often described than actually present. Uhthoff regards it as symptomatic of distension of the vaginal space and perineuritis. Retinal hæmorrhage, retinitis, etc., are rare, and tubercle of the retina is probably always by extension from the choroid or optic nerve (v. Vol. II, pp. 617, 683).

Tubercle of the choroid occurs in 19 per cent. of cases according to the literature (cf. Uhthoff supra). The observations of Barlow— of 16 cases of choroidal tubercle 13 had tubercular meningitis, of Jessop —of 15 cases 14 had tubercular meningitis, and of Carpenter and Stephenson—of 36 cases 26 had tubercular meningitis, are extraordinarily high. The percentage of choroidal tubercle in general miliary tuberculosis is undoubtedly higher than in tubercular meningitis per se, viz. 42 per cent. in reported cases (Uhthoff). The histology of choroidal tubercle has already received attention (v. Vol. II, p. 462).

v. MICHEL.—Deutsches A. f. klin. Med., xxii, 1878 ; Münchener med. Woch., 1903. SATTLER.—A. f. O., xxiv, 3, 1878. GARLICK.—Med.-Chir. Trans., 1879. BRÜCKNER.— A. f. O., xxvi, 3, 1880. DEUTSCHMANN.—A. f. O., xxvii, 1, 1881. STEPHEN MACKENZIE.— T. O. S., ii, 1882. WARNER.—Lancet, 1882. BARLOW, MONEY.—Lancet, 1883. BOCK.— Virchow's Archiv, xci, 1883. EDMUNDS AND LAWFORD.—T. O. S., iii, 1883. WORTMANN.— Jahrb. f. Kinderheilk., 1883. LAWFORD.—T. O. S., v, 1885. HENOCH.—Vorlesungen über Kinderkrankheiten, Berlin, 1889. RIEDER.—Münchener med. Woch., 1889. REINHOLD.— Deutsches A. f. klin. Med., xlvii, 1891. ELSCHNIG.—A. f. O., xli, 2, 1895. MURATOW.—- Neurol. Centralbl., 1895. DANIEL, DAVIS.—Am. Med. Assoc., 1897. HINSELWOOD.— Glasgow Med. Jl., 1897. BUCHANAN.—Edin. Med. Jl., 1900. WARNICKE.—A. f. Ohrenheilk., xlviii, 1900. LITTEN.—Deutsche med. Woch., 1902. CHEVALLEREAU AND CHAILLOUS. —Rec. d'O., 1903. NACHT.—Z. f. A., xii, 1904. DE LIETO VOLLARO.—A. di Ott., xii, 1904. BARLOW.—In Allbutt's System of Medicine, vii, 1904. JESSOP.—Brit. Med. Jl., 1905 ; Internat. Congress, Lisbon, 1906. CARGILL AND MAYOU.—T. O. S., xxvi, 1906. CARPENTER

and Stephenson.—Trans. Soc. for Dis. of Children, i. Treacher Collins.—Internat. Congress, Lisbon, 1906. *Uhthoff.—In G.-S., xi, 2, 1907 (Bibliography). (*See* Vol. II, p. 467.)

Epidemic cerebro=spinal meningitis.—This disease, due to the *Meningococcus intracellularis* of Weichselbaum, shows great differences in the severity of the clinical symptoms, and the cases of posterior basic meningitis commonly so-called in England appear to be sporadic forms, though Still's coccus differs in minor details from Weichselbaum's. In 110 cases of epidemic cerebro-spinal meningitis Uhthoff found the following ocular complications : conjunctivitis twice, due to lagophthalmos; keratitis 3 times—once deep infiltration, once dendritic keratitis, once keratitis e lagophthalmo; optic neuritis 18 times, bilateral with one exception ; metastatic ophthalmia 4 times.

Conjunctivitis is considered rare by some authors, common by others. It is probably sometimes endogenous in origin, in other cases due to defective closure of the lids. The meningococcus has been found in the conjunctival sac in some of these cases (Axenfeld, Brons), but the difficulty in distinguishing it from the gonococcus and the *Micrococcus catarrhalis* must be borne in mind, as well as the fact that the organism has been found independently of meningitis.

Keratitis dendritica must be regarded as due to the herpes which so frequently accompanies cerebro-spinal meningitis (Axenfeld, Nieden, Aldrich, Heine, and others). Secondary infection may lead to severe ulceration of the cornea (Niemeyer, and others), or this may be caused by lagophthalmos (Ziemssen and Hess, Schirmer, Schmidt-Rimpler, and others). Deep infiltrates in the cornea were found bilaterally in one case by Uhthoff; one eye had metastatic ophthalmia.

The more recent investigations show that slight papillitis is not uncommon in cerebro-spinal meningitis (Heine, Friis, Wilbrand and Saenger, Randolph, Passiatore, Ormerod, Goeppert, Chance, Jochmann, Uhthoff). Typical choked disc is rare (Heine, Nieden, Uhthoff). Optic atrophy (Collins, Bull), plastic exudation around the chiasma (Kotsonopulos, Hoering, etc.), have been described. Heine observed a preretinal hæmorrhage, v. Ziemssen hæmorrhages near an atrophic disc. Randolph reported 11 cases of thrombosis of the central retinal vein in 40 cases of cerebro-spinal meningitis—an observation which, in the light of other observers' statistics, must be regarded as inaccurate. Of posterior basic meningitis Barlow and Lees record the condition of the optic discs in 42 cases. In 27 they were quite normal, in 8 decidedly pale but clear edged, and in 3 there was also pallor round the disc or along the course of the retinal vessels; in 4 there was deficient clearness of edge and 3 showed distinct optic neuritis. My own experience at the same hospital—the Children's Hospital, Great Ormond Street—leads me to regard these statistics as rather under-estimating the usual frequency, though there can be little doubt that optic neuritis is less common in this disease than in tubercular meningitis. Amaurosis is not uncommon in posterior basic meningitis and the light reflex of the pupil is often sluggish. Barlow and Lees record one case of pseudoglioma in this disease.

Anatomically, descending optic neuritis is not always found, as in

Uhthoff's case, in which there was dilatation of the vaginal space of the nerve with œdema of the nerve and papilla. Whether perineuritis is due to the presence of organisms or to toxins alone is uncertain; the cocci may be absent in such cases in pneumococcic meningitis (Axenfeld). On the other hand micro-organisms may be plentiful in the sheaths in purulent meningitis without causing papillitis; even abscess formation may occur in the nerve without setting up intra-ocular inflammation (v. Hofmann). Very early optic neuritis is attributed by Westenhoeffer to hæmatogenous propagation. Direct continuity of inflammation from the meninges to the nerve sheaths is shown by the observations of Cheatham and Radmann.

Metastatic endophthalmitis is relatively common in cerebro-spinal meningitis (Heine 5 in 100 cases, Goeppert 3 in 44, Radmann 3 in 61, Curtius 7 in 200, Uhthoff 4 in 110); it occurs in about 4—5 per cent. of cases. A large number of cases have been reported (see Uhthoff). Clinically there is little external inflammation, and the yellow reflex from the pupil (amaurotic cat's eye) is the most striking feature. It is probable that some pneumococcic cases have been included; staphylococci and streptococci, on the other hand, tend to produce purulent panophthalmitis. The latter is rare in epidemic cerebro-spinal meningitis (cf. Knapp, Markusy, Uhthoff). Slight cases, showing only iritis or iridochoroiditis, are reported (Knapp, Heine, Schweitzer, Bull, Senator), but are rare; they are commoner in recurrent fever (Uhthoff). The complication is usually unilateral, though bilateral cases are recorded (Knapp, Kreitmair, Seggel, and others); both eyes are then generally affected simultaneously.

Anatomical investigations show that the complication may commence either as a metastatic retinitis or choroiditis (Uhthoff, Axenfeld, Knapp, Berthold, Rudnew and Burzew, Oeller, Saltini, Weeks, Wintersteiner), but the latter seems to be the commoner. Foster records two cases of direct transmission from the optic nerve, but this mode must be extremely rare. The optic nerves may show no inflammatory changes (Oeller, Axenfeld, Uhthoff, and others). Direct transmission from the nasal cavity is improbable. Blood transmission is most likely, and is supported by the observations of Martini and Rohde, who found meningococci in the blood, even before the occurrence of meningitic symptoms.

Meningococci have only rarely been found in the eye (Axenfeld, Wintersteiner, Stephenson); they probably rapidly degenerate here and disappear.

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Jl., 1903. Depène.—Allg. med. Central. Zeitung, 1903. Nacht.—Z. f. A., xii, 1904. Barlow and Lees.—In Allbutt's System of Med., vii, 1904. Foster.—Am. Jl. of Med. Sc., 1905. Heine.—Berliner med. Woch., 1905. Hildesheim.— Lancet, 1905. Morax.— Soc. d'O. de Paris, 1905. Nieden.— K. M. f. A., xlii, 1905. Radmann.—Deutsche med. Woch., 1905. Schottmüller.—Münch. med. Woch., 1905. *Uhthoff.—B. d. o. G., 1905; in G.-S., xi, 2, 1907 (Bibliography). Mayou.—R. L. O. H. Rep., xvi, 1906. Robinson. —Am. Jl. of Med. Sc., 1906. Brons.—K. M. f. A., xlv, 1907.

Otogenous purulent meningitis.—Purulent metastatic ophthalmia is extremely rare in otogenous meningitis, more so than in the epidemic cerebro-spinal form. Cases are reported by Nettleship, Cargill, Flemming, and others. They are due to general infection, not to direct transmission; organisms may, indeed, travel along the nerve sheath to the back of the globe without causing endophthalmitis (Axenfeld). It is possible that in some cases both the otitis and the endophthalmitis are subject to a common cause (Nettleship, Flemming).

Optic neuritis is rare in uncomplicated otitic purulent meningitis, and typical choked disc is probably always an expression of grave intra-cranial complication (cerebral abscess, subdural abscess, sinus thrombosis, etc.) Slight papillitis does, however, occur (Grunert and Schulze, Hansen, Macewen, and others). Pitt failed to find pathological changes in the optic nerves post-mortem. In the complicated cases optic neuritis of some degree of severity is the rule (Körner, Takabatake, Janson, and others). There is reason to accept the view that the swelling of the disc in pronounced papillitis in these cases is greater on the side of the lesion. Other ophthalmoscopic changes are very rare (Zaufal, v. Michel, Rampoldi).

v. Michel.—Deutsches A. f. klin. Med., xxii, 1878. Schultze.—Deutsches A. f. klin. Med., xxv, 1880. Andrews.— Med. Rec., 1883. Nettleship.—T. O. S., v, 1885. Kipp.— Z. f. Ohrenheilk, xv, 1886. Rampoldi.—Ann. di Ott., xviii, 1889. Pitt.—Brit. Med. Jl., 1890. Jansen.—A. f. Ohrenheilk., xxv, xxvi, 1893. Lane.—Brit. Med. Jl., 1893. Elschnig. —A. f. O., xli, 2, 1895. Rakowicz.—K. M. f. A., xxxiii, 1895. Pooley.— Med. Rec., 1896. Ostmann.—A. f. O., xliii, 1, 1897. Cargill.—T. O. S., xviii, 1898. Percy Flemming.— T. O. S., xx, 1900. Hansen.—A. f. Ohrenheilk., xxxv, 1901. Grunert and Schulze.— A. f. Ohrenheilk., liv, 1902. Schulze.—A. f. Ohrenheilk., lvii, lviii, 1903. Takabatake.— Z. f. Ohrenheilk., xlv, 1903. *Uhthoff.—In G.-S., xi, 2, 1907 (Bibliography).

Other forms of meningitis.—Optic neuritis occurs sometimes associated with the meningitis of fevers, e.g. typhoid, pneumonia, influenza, scarlet fever, measles, erysipelas, etc. Metastatic endophthalmitis (Berthold, Shears, Scheffels, Sutphen, Silcock, and others), optic neuritis (Berthold, Manz, Felser, Davis, Higgens, and others), choked disc (C. Mayer, Deutschmann), retrobulbar and descending neuritis, etc., occur with purulent meningitis of unknown ætiology. The neuritis and consecutive atrophy of simple chronic meningitis is open to argument from the point of view of ætiology. Choked disc and retrobulbar neuritis occur in infiltration of the meninges by malignant growths.

Silcock.—T. O. S., xx, 1900. de Lieto Vollaro.—K. M. f. A., xli, 1903. Reis.— A. f. A., liii, 1905. *Uhthoff.—In G.-S., xi, 2, 1907 (Bibliography).

Hydrocephalus.—Uhthoff has collected 46 cases of choked disc in hydrocephalus; most of the patients were over 14 years of age. Choked disc is much less frequent in the hydrocephalus of children owing to the expansion of the skull under the increased intra-cranial

pressure. In cases where the cerebro-spinal fluid escapes, *e. g.* through the nose (Elliotson, St. Clair Thomson, Schwab and Green, and others), papillitis is absent. Probably most of these cases in adults are secondary in origin (serous meningitis). Slight optic neuritis or post-neuritic atrophy is less common : Uhthoff found it in 39 cases, of which 10 were confirmed by autopsy (Edmunds, Quincke, Oppenheim, Diller, Beck, Prince, Uhthoff, Bullard and Thomas). In 5 cases there was disease of the ear (Bramwell, Robson, Kausch, Emerson, Uhthoff); none were children. Two of the cases had escape of cerebro-spinal fluid from the nose (Leber, Nettleship). A case of Oppenheim's with bitemporal hemianopia is interesting, since the autopsy showed great pressure on the chiasma.

Primary optic atrophy was found by Uhthoff in 38 cases, of which 18 were examined post-mortem. About a third of the patients were under 10 years of age, half of these being less than 12 months. My own experience at the Children's Hospital, Great Ormond Street, is that primary atrophy is much commoner than optic neuritis in very young children with hydrocephalus. It is probable that the atrophy in most cases is due to pressure of the infundibulum upon the chiasma —a view supported by numerous autopsies. It is doubtful if descending neuritis occurs (*cf.* Leber, Purtscher). Priestley Smith reports a case of simple optic atrophy with outflow of cerebro-spinal fluid from the nose.

Other ophthalmoscopic changes are rare—absence of retinal vessels (Anton), retinal hæmorrhages (Kupferberg), star at the macula (Oppenheim), venous hyperæmia (Levi), diffuse retinitis. probably syphilitic (Neumann), etc. Malformation of the globe may be coincident—microphthalmia with persistent pupillary membrane (Bernheimer, Rochon-Duvigneaud), microphthalmia with cyst (Natanson).

Amaurosis without ophthalmoscopic abnormality is comparatively common (Uhthoff, 16 cases). Most of the cases are children under one year old. Relief of pressure by lumbar puncture, trephining. etc., may lead to improvement of vision. Mere pressure on the higher visual centres or actual degeneration may account for the blindness.

Exophthalmos is comparatively common (Uhthoff 12 cases). Nearly all the cases were young children, the condition being caused by malformation of the orbit. In other cases the eyes appear sunken.

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Jahrb. f. Kinderheilk., 1905. *Schwab and Green.—Am. Jl. of Med. Sc., 1905. Weber.—A. f. Psych., xxxix, 1905. *Uhthoff.—K. M. f. A., xliii, 1905 ; in G.-S., xi, 2, 1907 (Bibliography). Weisenburg and Thorington.—Am. Jl. of Med. Sc., 1906. Glynn.—Brit. Med. Jl., 1906. Rochon-Duvigneaud.—Ann. d'Oc., cxxxvii, 1907.

Affections of the Trigeminal Nerve.

The ophthalmic division of the fifth nerve supplies the eyeball and its surrounding parts with sensibility. Though the lacrymal secretomotor fibres run in the lacrymal nerve, a branch of the trigeminal, it is probable that they are derived from the facial (*cf.* Parsons). In addition to loss of sensibility of the cornea and conjunctiva paralysis of the trigeminal often causes ulceration of the cornea, a disorder which has long been attributed to the presence of trophic fibres. Irritative lesions of the nerve often cause an herpetic eruption in the superficial course of the fibres.

Parsons.—R. L. O. H. Rep., xv, 2, 1902.

Herpes ophthalmicus.—The branches usually affected in this disease are the supra-orbital, the supra-trochlear, the infra-trochlear and lacrymal ; less frequently the nasal. Scars in the skin, which may be keloid (Holmes Spicer, Parsons), are always caused. Generally there is cutaneous, conjunctival and corneal anæsthesia, associated with neuralgic pain. Sometimes there is cutaneous anæsthesia dolorosa, seldom hyperæsthesia. The disease is rarely bilateral (Laillier, Samelsohn, Schiess-Gemuseus, Jaclard, Douglas) — according to Hutchinson never, and very rarely (Berger, Nieden, Kaposi, Tilbury Fox, Kopp, and others) occurs twice in the same nerve area (Jacksch). In more than half the cases lesions of the globe occur (Hybord, 44 out of 98 cases ; Pacton, 89 out of 126 ; Koch, 46 out of 80). Hutchinson states that it is not affected unless the naso-ciliary branch is involved ; though partially true this law is not without exceptions (Jacksch, Vernon, Coppez, Cohn), nor is the globe always affected when the naso-ciliary branch is involved (Douglas, Jeffries, Moers, Wadsworth).

There is usually hyperæmia of the conjunctiva and often pronounced conjunctivitis. There may be a crop of vesicles on the conjunctiva (Scriven, Sichel Jr., Arlt, Lagarde).

The corneal complications resemble those of herpes febrilis. The acute vesicular form is less often seen than in herpes febrilis. Minute ulcers may form, enlarge, and fuse, a considerable area of the cornea becoming denuded of epithelium, and this area continues to increase, showing a crenated border. In other cases a typical dendritic ulcer is formed. Grey striæ extend in one or more directions from the minute ulcer, grow longer and throw out lateral branches, finally breaking down. The ulcers are superficial and never extend in depth unless they become infected, when an hypopyon ulcer may result. They are extremely chronic and subject to relapse. Much more frequently than in febrile herpes they are accompanied by deep infiltration of the cornea, and this not infrequently occurs in herpes ophthalmicus without ulceration. In some cases typical neuroparalytic keratitis (q. v.) is set up. That the disease of the cornea may commence with

vesicles is shown by the cases of Horner, Kendall, Berlin, Wyss, and others.   Cases of deep infiltration have been published by Wangler, Wilbrand and Saenger, Sulzer, Cohn, and others.   Though neuro-paralytic keratitis is rare in this disease it would appear certain that it occasionally happens (Ginsberg, Kroll, Hybord).   Iritis (Hutchinson, Horstmann, Machek, Wyss), or more frequently iridocyclitis with precipitates ("k. p.") on the back of the cornea (Pacton, Sattler, Noyes, Koch, Cohn, and others), occur more often in ophthalmic than in febrile herpes.   Decreased intra-ocular pressure has often been des-cribed as characteristic (Horner, Goldzieher, Horstmann, and others).   Optic neuritis, retinitis, and choroiditis are rare.

Herpes ophthalmicus may occur at any age, though it is rare in early childhood (Pacton, Hybord) ; males are more often attacked than females (Jaksch, Koch, Laqueur, Hybord, Pacton).   The disease is not very common, but is said to show a tendency to epidemic out-breaks (Kaposi, Head, Parsons).   The not infrequent association with influenza, prolonged administration of arsenic, etc., points to a toxic origin.

Allied to the typical forms of herpetic keratitis are other cases which are not vesicular but which generally follow some febrile attack, and pursue a course strikingly similar to true herpes.   Such is the superficial punctate keratitis of Fuchs (cf. Wehrli), to which group belong cases such as I have described elsewhere showing slight varia-tions.   Other parerpetic conditions are idiopathic filamentary keratitis, cases which show an extreme tendency to desquamation of the corneal epithelium (Grandclément, Bronner, Menzies, Bartels, Franke, Kauff-mann), etc.

The pathology of these herpetic and parerpetic conditions is of much interest.   As long ago as 1862 herpes zoster was attributed to lesions of the posterior root ganglia (v. Bärensprung).   Wyss (1871), Sattler (1875), and Kaposi found hæmorrhage and infiltration of the Gasserian ganglion in herpes ophthalmicus.   The subject was attacked and exhaustively investigated by Campbell and Head in 1900.   They examined 17 cases of herpes zoster of varying duration, from a few days to one and a half years after the eruption.   In all the acutest cases hæmorrhages into the ganglion were found, usually small but surrounded by a considerable amount of inflammatory exudation. The ganglion cells were destroyed to a variable extent, and in the later cases parts of the ganglion were markedly sclerosed.   Three cases of zoster within the territory of the trigeminal were investigated, and in all changes were discovered in the Gasserian ganglion.   Other observers have noted similar but less marked changes in posterior root ganglia above and below the one specially connected with the nerve area involved.   The analogy to acute anterior poliomyelitis has been repeatedly pointed out, but this analogy has perhaps been pressed too far.

There is therefore good evidence to believe that ophthalmic herpes is dependent upon definite lesions in the Gasserian ganglion, which is morphologically a dorsal root ganglion.   It is probable that the pathology of the parerpetic cases is fundamentally the same, different

as are the clinical features in certain details. The frequency of a febrile onset is striking, and though herpes febrilis does not manifest the same accuracy of distribution according to nerve supply that is found in herpes zoster, it is yet probable that the causal lesion should be referred to the peripheral sensory nerves, and most likely to their ganglia rather than to their terminations. Herpes zoster is the outcome of an intense and concentrated attack upon certain dorsal root ganglia. Herpes febrilis and the parerpetic affections of the cornea (and possibly of other parts of the body) may reasonably be regarded as a less acute, more widely diffused attack of a similar nature. In the parerpetic forms we must predicate less destruction of tissue and abolition of function, but finer localisation, often combined with wider diffusion, of partial derangement of tissue and disorder rather than demolition of function (Parsons).

Granting the accuracy of these views as to the site of the lesion there remains the extremely difficult task of explaining its manifestations. We are accustomed to regard sensory nerves as conveying only afferent impulses, and it is difficult to imagine how even an inflammatory block in the course of the nerve can produce the pathological changes in structure at its distant terminations. The trophic control of the nerve-fibres themselves by the dorsal root ganglia is a well-established fact. The trophic control of the tissues supplied by the fibres, early invoked to account for the phenomena of neuropathic keratitis, is more open to doubt. It is probable that neuropathic keratitis (q. v.) is due neither to abrogation of trophic influence nor to this cause assisted by loss of sensation, invasion of pathogenic organisms, etc. There is reason to believe that in all those cases in which the cornea becomes ulcerated after division of the fifth nerve the cut ends of the nerve are subjected to some abnormal irritation, either by blood-clot, pus, or other agent. This view fits in well with the manifestations of herpes and parerpetic affections. Such irritation can only make itself felt at the distal end of the nerve by the transmission of impulses along the nerve in the reverse direction to the normal afferent impulses or to the transmission of deleterious agents along the nerve.

There is some experimental evidence of the transmission of impulses along sensory nerves in the opposite direction to that of the normal impulses. In 1876 Stricker showed that vaso-dilatation of the hind limb of the dog occurred when the peripheral ends of the divided posterior roots of the sixth and seventh lumbar nerves were stimulated. In 1900, Bayliss, contrary to his expectation, confirmed these results, and subsequently adduced other examples of a similar nature. He called these efferent impulses occurring in afferent nerves *antidromic impulses*. Further reference is made to this matter in dealing with neuroparalytic keratitis (q. v.).

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A. f. Heilkunde, xii, 1871. SULZER.—Ann. d'Oc., cxix, 1898. GINSBERG.—C. f. A., xix, 1895. KROLL.—C. f. A., vi, 1882. MACHEK.—A. f. A., xxxi, 1895. SATTLER.—Wiener med. Presse, 1875; Wiener med. Woch., 1889. NOYES.—T. Am. O. S., 1873. HEAD.—In Allbutt's System of Med., viii, 1901. WEHRLI.—K. M. f. A., xliv, 1906. GRANDCLÉMENT.— Internat. Congress, Paris, 1888. BRONNER.—T. O. S., ix, 1889. MENZIES.—Ophth. Rev., xxi, 1902. BARTELS.—Münchener med. Woch., 1904. FRANKE.—K. M. f. A., xliv, 1906. KAUFFMANN.—K. M. f. A., xlv, 1907. GALEZOWSKI AND BEAUVOIS.—Rec. d'O., 1906. HOLMES SPICER.—Ophth. Rev., xxvi, 1907. JESSOP.—T. O. S., vi, 1886. v. BÄRENSPRUNG. —Charité Annalen. xi. SATTLER.—Berliner klin. Woch., 1875. BAYLISS.—Jl. of Physiology xxvi, xxviii. *CAMPBELL AND HEAD.—Brain, 1900. *WILBRAND AND SAENGER.—Die Neurologie des Auges, ii, 1901 (Bibliography). LAUBER.—A. f. O., lv, 1903. CASPAR.— A. f. A., xlviii, 1903. ZENTMAYER.—Amer. Med., 1903. PRETORI.—A. f. A., lvii, 1907. *PARSONS.—Lancet, 1907.

Neuroparalytic keratitis is a comparatively rare disease; it accompanies paralysis or paresis of the trigeminal nerve or of its ophthalmic branch, but is by no means present in all such cases. Of late years this fact has been conspicuously demonstrated by the many cases of extirpation of the Gasserian ganglion for trigeminal neuralgia at the hands of Krause, Sir Victor Horsley, and other surgeons. If due care is exercised only a small proportion of these cases develop corneal complications. The disease is, indeed, the more mysterious on this account, for in most cases it is impossible to discover any adequate cause for it. Other morbid conditions which lead to paralysis of the fifth nerve or its ophthalmic branch may induce neuropathic keratitis. The seat of the lesion may be in the sphenoidal fissure or cavernous sinus, peripheral to the ganglion, involving it, or proximal to it; it may even be in the brain involving the central connections of the nerve. The operative removal of the Gasserian ganglion, however, is best suited to provide evidence of the true *rationale* of the disease, for it has all that accuracy of localisation which pertains to an experiment and is so often open to doubt in the manifestations of disease.

In most cases in which neuroparalytic keratitis follows extirpation of the ganglion it commences during the first days after the operation. There are a few cases in which corneal anæsthesia due to disease has resulted in this form of keratitis only after an interval of months or years (up to four years). In a typical example the cornea becomes dull and the epithelium is thrown off, first at the centre, then more and more peripherally, until eventually there is only a narrow rim, from two to three millimetres broad at the margin. There can be little doubt that this vulnerability of the epithelium is the characteristic feature of the disease. The subsequent increase in the opacification, development of hypopyon, perforation, etc., are attributable to the invasion of the substantia propria by pyogenic organisms, now rendered possible by the removal of the first line of defence. The rapidity of the destruction of the deeper layers of the cornea, which is frequently noticeable, indicates that the resistance of the tissues is diminished, but affords no indubitable evidence that this is due to the affection of the nerve. There is, of course, no pain, but there is conjunctival and often ciliary injection. The absence of lacrymation is due to the block in the afferent limb of the reflex arc, and has no bearing upon the question of the true secreto-motor supply of the gland, whether derived from the fifth or seventh nerve.

The lesions of the cornea in disease of the trigeminal nerve may be slight superficial or deep infiltration, or both combined with or without ulceration. If an erosion of the epithelium occurs and it becomes infected hypopyon ulcer rapidly ensues in most cases and may lead to perforation of the cornea, panophthalmitis, and loss of the eye (cf. Wilbrand and Saenger).

Many theories of neuroparalytic keratitis have been advanced, for the subject early attracted attention owing to the results of experimental division of the fifth nerve in animals. Only brief reference can be made to them here; they have been exhaustively reviewed by Wilbrand and Saenger.

The *trophic theory*, enunciated by Magendie (1824), ascribed the disease to the abolition of special trophic nerve-fibres running in the nerve. These fibres under normal conditions control the nutrition of the cornea. Similar fibres were supposed to run in other nerves, their destruction accounting for the development of bedsores and other " trophic " lesions. It is noteworthy that Gaule opposed the theory on the ground that no centrifugal fibres were distributed to the cornea, but the chief fact which militated against the theory was the possibility of warding off the disease by protecting the eye from external injury. Hence arose the *trophic-traumatic theory*, which asserted the necessity of injury in addition to diminution in resistance of the tissues brought about by the destruction of the trophic fibres. Wilbrand and Saenger have collected the clinical cases bearing upon this view. They include cases of paralysis of the facial nerve, causing exposure of the eye, in addition to paralysis of the trigeminal; in many of these neuroparalytic keratitis ensued, but in many others it was absent. Similarly there are many cases in which ptosis failed to protect the eye sufficiently to ward off the disease, though it frequently was absent in others of the same group. Other cases include lesions of the trigeminal with retained sensibility or even hyperæsthesia of the cornea, yet accompanied by keratitis, and others with complete corneal anæsthesia yet no keratitis. Cases of delayed keratitis neuroparalytica are recorded (Francke, Hirschberg, Jany, Pufahl, Nieden).

The *vasomotor theory* was brought forward by Schiff (1867), attributing the inflammation to dilatation of the blood-vessels owing to paralysis of the vasomotor nerves; it was supported by Spalitta. In a later modification the *vasomotor-traumatic theory* admitted the influence of external injury; the theory is supported on the clinical side by Seydel.

The pure *traumatic theory* ascribed the unusual effect of injury to the insensibility of the cornea. Snellen showed that the disease could be warded off by sewing together the lids. This aspect of the question was very thoroughly investigated experimentally and microscopically by Senftleben under Cohnheim's direction and by v. Gudden.

The *xerotic theory* (Feuer) also eliminated the influence of trophic fibres, invoking the deleterious effect of drying of the cornea, owing to diminished frequency of blinking, etc. A strong argument against the blinking factor is the symmetrical blinking which occurs in man, whereas the paralysis is almost invariably unilateral. The theory is supported by E. v. Hippel and Ollendorf.

Bacterial invasion was invoked as the cause of the disease in the *mycotic theory* (Eberth).   This theory has recently received support from work by Morriston Davies and Hall ; the special organism which they describe, however, much resembles the xerosis bacillus.

It will be observed that simple division of the fifth nerve affects the cornea in many ways.   The afferent sensory impulses are blocked, so that anæsthesia and analgesia result.   Hence slight injuries, foreign bodies, etc., are liable to pass unnoticed and produce deleterious effects. Accompanying the anæsthesia is the abolition of the reflex secretion of tears.   In lower animals there is also diminution in the frequency of blinking, absent in higher animals, except in bilateral paralysis, owing to the synergic activity of the two orbiculares.   The vaso-motor element is itself complex.   Vaso-constrictor fibres for the eye, derived from the cervical sympathetic, run near the Gasserian ganglion and join certain of the branches of the nerve.   Their division results in dilatation of the vessels.   It is probable that vaso-dilator fibres for some parts of the eye run in the trigeminal itself.   Their division results in vaso-constriction.   It is difficult to define the relative importance, if any, of these conflicting factors.   That the bacterial element is of importance in the later stages of neuroparalytic keratitis cannot be doubted, but it is unlikely that it explains the desquamation of the epithelium which is the characteristic feature of the disease, and is known to be caused only by such organisms (*e. g.* gonococcus) as are almost certainly absent.

As already mentioned it has been shown, chiefly on the evidence brought forward by Head and Campbell, that the causal lesion in herpes ophthalmicus is situated in the Gasserion ganglion.   In the absence of known efferent channels in the parts of the fifth nerve under consideration it was necessary to have recourse to antidromic impulses travelling centrifugally along the " afferent " fibres, basing the explanation upon the proved occurrence of such impulses under certain conditions by Bayliss.   Head has now carried his researches upon the sensory nervous system a vast stride forwards.   He has chosen his coadjutors, Rivers, Sherren, and Thompson, with the same acumen that he has displayed in devising the details of the research, for they are severally specially qualified to attack the problems allotted to them. Commencing with an investigation of lesions of the peripheral nerves, Head has arrived at the conclusion that three types of sensibility may be distinguished, called respectively deep, protopathic, and epicritic, and that the corresponding impulses travel to the central nervous system by three distinct sets of fibres.   Deep sensibility persists after destruction of all cutaneous afferent fibres.   The impulses travel in motor nerves (thus confirming previous work of Sherrington) as far as the separation of the anterior and posterior spinal roots, when they pass into the posterior roots and join the rest of the afferent system.   This form of sensibility includes deep pressure, movements of muscles, localisation of pressure, and recognition of the extent and direction of passive movements in joints.   Loss of protopathic sensibility abolishes cutaneous pain, sensations of heat above 45° C., and sensations of cold below 20° C.   Loss of epicritic sensibility

abolishes recognition of light touch, discrimination of compass points, appreciation of differences in size, and discrimination of intermediate degrees of temperature from 25° to 40° C. After division of a sensory nerve protopathic sensibility is restored in about six weeks if the ends of the nerve are placed in apposition. Cutaneous sores which may have occurred in the area affected from injury, etc., show little or no tendency to heal until protopathic sensibility is restored, when healing rapidly ensues. Epicritic sensibility requires much longer for restoration, and the time varies with the position of the affected part. The areas of protopathic and epicritic sensibility are not identical. In any peripheral nerve the distribution of the protopathic fibres usually overlaps greatly the area supplied by the fibres of the adjacent nerves, whilst the distribution of the epicritic fibres in the larger peripheral nerves, such as the median and ulnar, overlaps only slightly. It is found for certain peripheral nerves or nerve groups—such as the median, ulnar, or pre-axial nerves of the arm—that each of these forms a unit of the epicritic system, whilst the protopathic unit must be sought in one or more posterior nerve roots, for the nearer the lesion is situated to the posterior roots the more extensive and definite is the loss of protopathic sensibility. Similarly, the more nearly the injury divides one of the nerve groups the more definite and extensive is the epicritic loss.

　　Lesions of the spinal cord show that there is a complete redistribution of afferent impulses, so that peripheral impulses are transmuted into those of the secondary level of the afferent nervous system. This transmutation and recombination takes place on the same side as that by which the impulses enter the cord. The secondary paths for sensory impulses then cross with greater or less rapidity, so that ultimately all except those subserving the sense of passive position and movement and tactile discrimination have passed to the opposite side within the limits of the spinal cord. Even these sensory impulses cross after reaching the nuclei of the posterior columns. At the same time within the cord afferent impulses become separated into sensory and non-sensory. Of the latter many pass up in the secondary system of the direct cerebellar tract to reach the cerebellum.

　　It has been mentioned that skin lesions in an analgesic area show little or no tendency to heal until restoration of protopathic sensibility has taken place. There is further evidence that these peripheral lesions are in some manner under the control of the protopathic system. Head has previously shown that there is a close correspondence between the distribution of the tenderness caused by irritation through visceral disease of the segments within the cord and the areas marked out on the skin by the eruption of herpes zoster. He has also shown that herpes zoster is due in most cases to acute inflammation of a posterior root ganglion, whereas the tender areas in visceral disease are due to irritation of intra-medullary segments. But, though all the cells and the fibres peculiar to them must be affected by the profound inflammation of the ganglion, one system only, so far as we know, can produce antidromic effects upon the skin, and these are the fibres shown by Bayliss to have their cells of origin in the ganglion of the

posterior root.   Head and Bayliss have now shown that these anti-
dromic fibres are capable of excitation in the divided nerve of a cat
five weeks after it has been reunited to the central nervous system.
Hence, it may be considered proved that the power of producing
changes in the skin of the periphery is a function of fibres which run
in the protopathic system.

Without discussing the bearing of these researches upon the afferent
impulses in the trigeminal nerve and their redistribution within the
central nervous system, for which the material is indeed as yet insuffi-
cient, it is clear that they have an important bearing on the pathology
of neuroparalytic keratitis.   Whether such so-called trophic lesions
can be caused by the abolition of antidromic impulses alone—*i. e.*
whether antidromic trophic impulses exist as a physiological phe-
nomenon—is a question which may be set aside for the present, though
the evidence in favour of such impulses is undoubtedly increased.
What seems to be certain is that abnormal stimulation of the proto-
pathic fibres in or near the ganglion produces antidromic impulses
which have a deleterious effect upon the nutrition of the peripheral
organs.   The researches, in fact, bring forward strong evidence in
support of a theory already suggested by Wilbrand and Saenger—viz.
that neuroparalytic keratitis is due to irritation of the distal end of the
cut or diseased trigeminal nerve.   This theory explains better than
any other all the diverse clinical facts which have accumulated.   It
explains the absence of keratitis in those cases in which it does not
occur.   Neuroparalytic keratitis may occur in association with retained
corneal sensibility.   Here the afferent tract is still open, but an irrita-
tive lesion has set up abnormal antidromic impulses in the protopathic
system.   There may be hyperæsthesia of the cornea with keratitis.
Here not only is the afferent tract open but it is subject to abnormal
stimulation either at the periphery or at the site of the lesion, and
abnormal antidromic impulses are also set up.   There may be anæs-
thesia dolorosa.   This is due to irritation of the proximal end of the
cut or diseased nerve, whilst antidromic impulses are set up in the
distal section.

MAGENDIE.—Jl. de Physiol. exp., iv, 1824. * GAULE.—Centralbl. f. Physiol., 1891.
CLAUDE BERNARD.—Leçons, ii, 1858.   v. GRAEFE.—A. f. O., i, 1854.   SCHIFF.—Z. f.
rationelle Med., 1867 ; A. des Sc. physiques, 1886.   SAMUEL.—Die trophischen Nerven,
Leipzig, 1860.   BÜTTNER.—Z. f. rat. Med., xv, 1862.   SPALITTA.—Arch. di Ott., ii, 1894.
OLLENDORF.—A. f. O., xlix, 1900.   SEYDEL.—A. f. O., xlviii, 1, 1899.   SNELLEN.—Holländ.
Beiträge f. Natur- u. Heilkunde, 1857.   SENFTLEBEN.—Virchow's Archiv, lxv, 1875.   v.
GUDDEN.—Neurol. Centralbl., iii.   FEUER.—Wiener med. Presse, 1877.   E. v. HIPPEL.—
A. f. O., xxxv, 3, 1889 ; xlvii, 1, 1899.   EBERTH.—Centralbl. f. med. Wissenschaft, 1871.
TURNER.—Brit. Med. Jl., 1895.   KRAUSE.—Die Neuralgie des Trigeminus, Leipzig, 1896.
FLEMMING.—Lancet, 1898.   BAYLISS.—Jl. of Physiol., xxvi, 1901 ; xxviii, 1902.   HEAD,
RIVERS, AND SHERREN, HEAD AND SHERREN.—Brain, xxviii, 1905.   HEAD AND THOMPSON.
—Brain, xxix, 1906.   MORRISTON DAVIES.—Brain, xxx, 1907 (Bibliography of Extirpation
of Gasserian Ganglion).   *WILBRAND AND SAENGER.—Die Neurologie des Auges, ii,
Wiesbaden, 1901 (Bibliography).   LAWFORD.—T. O. S., xxvii, 1907.   WEISS, JR.—K. M. f. A.,
xlv, 1907.   PARSONS.—Lancet, 1907.   STEINDORFF.—C. f. A., xxv, 1901.   METTEY.—A. d'O.,
xxiii, 1903.   ALBRAND.—Wiener klin. Rundschau, 1903.   *DAVIES AND HALL.—Brit. Med.
Jl., 1908.

CHAPTER XXVIII

HEREDITY

THE influence of heredity in many diseases of the eye has been frequently mentioned in the course of this work. Much attention has recently been devoted to the subject, and although the time is not yet ripe for dogmatic statements there are indications that the general principles underlying the inheritance of disease are slowly emerging from the complex mass of facts. In discussing the question it is well to group separately those cases in which manifest disease of the parent is transmitted to the offspring, as for example in the case of syphilis. The transmission of infectious disease to the offspring is not an example of true heredity, though reasons have been given to show that it accounts for some, perhaps many, cases of congenital anomaly (v. Vol. III, p. 771). Further, sporadic manifestations of congenital anomalies, even in the absence of clear signs of disease in the parents, are not necessarily due to hereditary influence. At any rate, their causation is too obscure to afford much assistance in elucidating the problem of heredity, and they are better segregated in a group apart.

A distinction has been made by some authors, e.g. Bollinger, between *direct* inheritance from parent to children, and *indirect* inheritance from grandparents, uncles, or aunts. *Collateral* inheritance is the term used to indicate the occurrence of the same anomaly in several members of the children of one parentage.

The individual children of one parentage are conveniently termed *siblings* (Karl Pearson), the whole group being called a *childship* (Stainer). Diseases which show a marked tendency to occur amongst the siblings of a childship, but have not hitherto been proved to show much tendency to transmission to succeeding generations, are conveniently called *familial* diseases. Examples of this group are found in nodular and reticular opacities of the cornea, amaurotic family idiocy, etc. They are allied in some respects to sporadic congenital anomalies, and should be treated similarly as a separate group.

The possibility of the transmission of acquired characteristics has been much discussed by biologists, and has been strenuously denied by His, Kölliker, Weissmann, Ziegler, and others. Some evidence bearing upon the question is derived from ocular conditions. Darwin mentions a man who lost his left eye from panophthalmitis fifteen years before his marriage ; he had two sons with left congenital microphthalmia.

Fuchs records the case of a man whose right eye was blinded by iridocyclitis; he had a son with right microphthalmia. Somewhat similar cases are reported by Perlia and Münden. Brown-Séquard removed one eye from guinea-pigs, and found that some of the offspring had microphthalmia or other ocular abnormalities. Deutschmann inoculated a male rabbit with tubercle in the iris; of six offsprings one had a shrunken globe and another disseminated choroiditis and optic atrophy. Similar experiments were made by Samelsohn with positive results. Mulder, during six years, removed the right eye in rabbits, but none of the 200 rabbits of the family operated upon showed any ocular abnormality. He also inoculated the anterior chamber of two rabbits with tuberculous iris; there was but little reaction, yet the three offspring all had opacities at the posterior pole of the lens, which Mulder attributed to lenticonus posterior.

The question whether the earlier or later children of a childship are most likely to be affected by inherited disease has been investigated by Laqueur. In forty-eight families, consisting of 244 children, 79, or one third, showed inherited ocular disease, two thirds escaping. The affected members were—first born, 25 per cent.; second born, 13 per cent.; third born, 36 per cent.; fourth born, 51 per cent.; fifth born, 37 per cent.; sixth born, 50 per cent.; seventh born, 50 per cent. The percentages are obviously unreliable amongst the later born, owing to the small numbers upon which they are founded.

The deleterious influence of consanguineous marriages is shown in several ocular diseases, e.g. retinitis pigmentosa. Too much stress has, however, been laid upon it. There seems little doubt that it has no effect per se, but only by virtue of the probability of the same weakness occurring in both members of the same stock, so that their union in marriage causes summation.

Dupuy.—Internat. Congress, 1876. Brown-Séquard.—Gaz. méd. de Paris, 1880; Comptes rendus, 1882. Deutschmann.—K. M. f. A., xviii, 1880. Samelsohn.—Centralbl. f. d. med. Wissensch., 1880. Fuchs.—Die Ursachen u. die Verhütung der Blindheit, Wiesbaden, 1885. Ziegler.—In Ziegler and Nauwerck, Beiträge zur path. Anat., i, 1886; iv, 1889 (Bibliography). Zepler.—Dissertation, Breslau, 1886. Perlia.—K. M. f. A., xxv, 1887. Nolte.—Dissertation, Marburg, 1896. Laqueur.—Z. f. prakt., Aerzte, 1897. Mulder.—K. M. f. A., xxxv, 1897. *Thomson.—Heredity, London, 1908.

The recent revival of interest in the subject of heredity is largely due to the re-discovery of the principles described by Mendel in 1865 by de Vries and others in 1900. The following exposition of the Mendelian theory consists of verbatim excerpts from a paper by R. C. Punnett, read before the Epidemiological Section of the Royal Society of Medicine (1908).

"As a simple illustration we may take a well-known case among poultry, that of the blue Andalusian fowl. It is a bird which has long been known to possess an inconvenient peculiarity: it will not breed true. It always throws "wasters" of two sorts: blacks, and whites marked with some black splashes. There are, therefore, three kinds of Andalusians, and consequently six possible types of mating among these three varieties. With regard to the results of these types of mating, careful experiment has brought out the following facts:

Blue   × Blue gives Blacks, Blues, and Whites, in the ratio 1 : 2 : 1.
Blue   × Black   ,,   Blacks and Blues in equal numbers.
Blue   × White   ,,   Blues and Whites   ,,   ,,
Black × Black   ,,   Blacks only.
White × White   ,,   Whites only.
Black × White   ,,   Blues only.

" We are dealing here with a case in which every possible form of mating has been carried out, and some of the results at first sight seem paradoxical. Thus, for instance, the blacks always breed true whatever their ancestry may have been ; and the same holds good for the whites. The white that is produced by two blues, themselves the product of mating blue with blue over many generations, breeds as true to whiteness as the white of pure white ancestry. A black is pure for blackness, and a white is pure for whiteness whatever the ancestry of the bird may have been. Again, it seems at first sight incongruous that the mating of black with white should give just twice as many blues as two blues mated together.

" But the theory of heredity first propounded by Gregor Mendel enables us to summarise all these results in a very simple and beautiful way. Briefly it is as follows : We are dealing with an alternative pair of characters, blackness and whiteness. Every germ-cell or gamete, whether ovum or spermatozoon, bears a representative of this pair. But it can bear only one representative, viz. *either* blackness *or* whiteness. Hence, for this pair of characters there are two, and only two, types of gamete—"black" gametes and "white" gametes. When a black gamete meets a black the result is a black bird ; when a white meets a white the result is a white bird. But when a white meets a black the resulting zygote contains the representatives or factors for both blackness and whiteness and develops into a blue bird. Now we must suppose that the gametic representative of a character, the factor, is an unsplittable entity so far as inheritance is concerned. The zygote, being formed by two gametes, must contain two factors. It is a double structure, and when it comes to form gametes these single structures are produced by the separation of the two factors present in any zygotic cell. The factors representing the characters are said to *segregate* from one another in the process. In a zygote produced by the union of similar gametes, the segregation is between like factors, and all the gametes produced are alike. But a zygote which has been formed by two dissimilar gametes, each bearing one of the factors corresponding to a pair of characters, must, on forming gametes, give rise to gametes of two sorts, and must give rise to them in equal numbers. On this simple hypothesis is afforded a ready explanation of the various experimental facts given above. A blue hen is producing equal numbers of "black" and "white" eggs—let us say $2n$ of each. To fertilise these eggs are brought large numbers of spermatozoa of the two sorts, black and white, in equal numbers. Every black egg, then, has an equal chance of being fertilised by a black or a white spermatozoon. In the former case it will form a black, and in the latter a blue, bird. From our $2n$ black eggs we shall obtain n black and n blue birds. Similarly, from our $2n$ white eggs we shall get n blue and n white birds. That is to say, the mating of blue with blue must, on the assumption

of the purity of the gametes, give black, blue, and white birds in the ratio 1 : 2 : 1.

" We may now put in a more general form what we have learned from this and similar cases. The characters of plants and animals may in many cases be regarded as existing in alternative pairs. Corresponding to each member of such a pair is something representing it which may be carried by the gamete. These factors which the gamete carries are the channel by which the qualities of the parent are transmitted to the offspring. Every gamete contains one, and only one, of the factors corresponding to a given pair of characters, i. e. is pure for that character. For any given pair of characters, therefore, there can be two, and only two, classes of gametes—those pure for one member of the pair and those pure for the other member of the pair. But there can be three different kinds of zygote, for each zygote is formed by the union of two gametes ; and since two kinds of gamete exist it is obvious that three kinds of union among them are possible. Two gametes, each pure for one member of the alternative pair of characters, may unite ; or two gametes, each pure for the other member of the pair, may unite ; or thirdly, two unlike gametes may unite. The zygote so formed contains representatives of each member of the pair and is known as a *heterozygote* (hybrid), whereas zygotes containing representatives of but one member of the pair are termed *homozygotes*. Like the homozygotes, the heterozygote produces pure gametes ; only it produces equal numbers of the two kinds instead of producing all of one kind. In this lies the explanation of the fact that hybrids mated together produce a definite proportion of the pure forms, which subsequently breed true without ever giving a hint of their mixed ancestry.

" In the simplest cases, such as those of the Andalusian fowl, we are dealing with but a single pair of characters, in so far as the gametes are concerned, and we are able to distinguish in appearance the birds arising from the three forms of zygote that these gametes can form. But in a large number of cases it is not possible to distinguish the hybrid from one of the parents. Rosecomb bantams exist in two forms, white and black. Each form breeds true, but when the two are crossed the hybrids all resemble the black parent. The zygote which contains a single dose of blackness grows up into a bird which is as black as the pure black containing a double dose of blackness—a point of difference to the
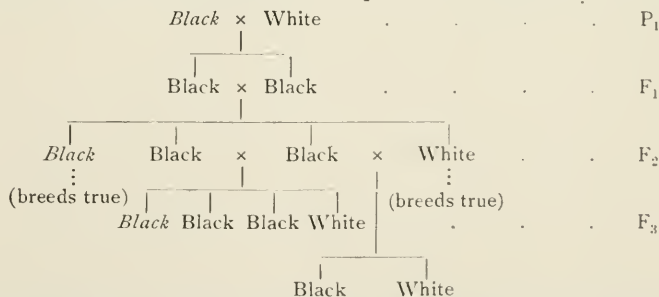
Scheme illustrating inheritance in Rosecomb bantams. Homozygous blacks in italics to distinguish from heterozygous. P_1 signifies parental generation, F_1 first filial generation, F_2 second filial generation, and so on.

Andalusian, where the zygote with only a single dose of blackness develops into the more or less intermediate blue. In cases such as this of the Rosecombs we use Mendel's terms, and speak of the character blackness as *dominant* to whiteness, which is said to be *recessive*. When the hybrids (F_1) are mated together they give, as we have already seen in the case of the Andalusians, one of each of the two homozygous forms for every two heterozygotes. But since black is dominant to white the heterozygotes are indistinguishable in appearance from the dominant homozygote, and this, the F_2 generation, consists visibly of three blacks to every white. The whites subsequently breed true, as do also the homozygous blacks when they are mated together. But if we wish to separate these homozygous blacks from the heterozygous we must devise some test. And the only test we know of at present is the test of breeding. All the gametes of a homozygous black contain the factor for blackness. Consequently, when such a bird is mated with a white all the offspring must be black. But a heterozygous black is giving off equal numbers of " black " and " white " gametes. Hence, when mated with a white it will form equal numbers of zygotes with and without a black factor, *i. e.* it will produce equal numbers of black and white birds, The test between the pure dominant and the dominant which carries the recessive character lies in crossing each with the recessive. The former produces only dominants, while the latter gives rise to equal numbers of dominants and of recessives. But whether the phenomenon of dominance is present or not, the essential feature of Mendel's discovery is unaffected, and this, of course, consists in the conception of the characters of living things as existing in alternative pairs, and of the purity of the gamete for either member of such a pair.

" *Dihybridism* is the term applied to cases in which the parents crossed differ from one another in two pairs of alternative characters. It was found by Mendel that in such cases the inheritance of each pair follows the same rule, but follows it independently. Tallness in the pea is dominant to dwarfness, and colour in the flowers is dominant to white. When, therefore, a tall coloured is crossed with a dwarf white all the offspring are tall plants with coloured flowers. In the next generation talls and dwarfs appear in the ratio 3 : 1, and coloureds and whites also appear in the ratio 3 : 1. Hence each tall plant has three times as many chances of being coloured as of being white. Similarly the dwarf coloureds must be three times as numerous as the dwarf whites. A moment's consideration will serve to show that the simplest

$$
\begin{array}{ccc}
\left.\begin{array}{l}\text{Tall}\\\text{Coloured}\end{array}\right\} & \times & \left\{\begin{array}{l}\text{Dwarf}\\\text{White}\end{array}\right.
\end{array}
$$

Tall
Coloured $\cdots\cdots\cdots\cdots\cdots\cdots\cdots\cdots\cdots\cdots\cdots\cdots\cdots\cdots$ F_1

Tall	Tall	Tall	Dwarf
Coloured… Coloured… Coloured… White …	Coloured… Coloured… Coloured… White …	Coloured… Coloured… Coloured… White …	Coloured… Coloured… Coloured… White …

expression which covers all these requirments is nine tall coloured, three tall whites, three dwarf coloured, and one dwarf white. And these are the proportions actually found by experiment in this and other cases. This is the $9 : 3 : 3 : 1$ ratio characteristic of cases of simple dihybridism, and we may state it in a more general form as follows : When two individuals are crossed which differ in two pairs of alternative characters, the F_2 generation consists of four classes, and out of every sixteen, nine on the average exhibit both dominants, three one of the dominants, and one of the recessives, three the other of the dominants and the other of the recessives, and one exhibits both recessives. The simple and orderly distribution of the characters to form this ratio may be taken as proof that each pair of characters, though obeying the same hereditary law, obeys it independently of the other.

" The distribution of two pairs of characters is not always so simple in appearance as in the case of the peas. The characters belonging to different pairs sometimes interact upon one another, and the way in which this comes about may best be explained by an example. A grey Belgian hare rabbit was crossed with an albino Angora. The progeny were all of the wild grey type. They were in-bred and produced in the next generation greys, blacks and albinos, the proportional numbers of the three kinds being $9 : 3 : 4$. The proportion of coloured rabbits to albinos is $3 : 1$, suggesting at once that colour and albinism are a pair of alternative characters, of which the former is dominant ; and among the coloured the ratio of greys to blacks $(9 : 3 = 3 : 1)$ points to greyness and blackness forming another pair of characters. If such is the case we ought to find among our sixteen rabbits twelve greys and four blacks. That we only find nine greys and three blacks is because one quarter of our sixteen rabbits must be albinos, lacking the colour factor which enables the particular colour present, whether grey or black, to declare itself. There must therefore be both grey albinos and black albinos, and this may be tested by mating an albino with a pure black. Since colour is dominant all the offspring will be coloured, but those albinos which carry the factor for greyness will give greys, and those without this factor will give only blacks ; and experiment has shown that this is the case. Albino rabbits may be compared to exposed but undeveloped negatives. The silver has undergone a change, but what the image is we cannot say until the developer is poured upon it. So with albino rabbits. By crossing with a black containing the factor which allows the colour to appear, we are, as it were, pouring on the developer, and the resultant colour, whether grey or black, tells us what manner of albino we had to deal with."

The most diverse characters in animals and plants have been shown to exhibit Mendelian inheritance. Examples of colour and size have been given in the quotations from Punnett's paper. More striking are such characters as the chemical constitution of granules in the endosperm of plants, their time of flowering, and the sterility of anthers. Even more remarkable is immunity to disease in wheat, for Biffen has succeeded in propagating a wheat on Mendelian principles which is immune to attacks of yellow rust. Ample evidence has been obtained

that the absence of a certain quality is often a Mendelian character, and such diseases as alcaptonuria and congenital night-blindness (*v.* p. 1400) are examples. Other diseases which have afforded examples of the Mendelian law are brachydactyly, diabetes insipidus, hereditary chorea, keratosis palmæ, congenital cataract (*v.* Vol. III, pp. 805, 1025), glaucoma, ectopia lentis, etc. In the examples given from Punnett the two factors in the zygote behave quite independently, but there are cases of what is called gametic coupling, in which there exists a tendency for factors to become definitely associated together. Other cases, again, show only incomplete coupling. There is some evidence that sex itself must, in certain cases, be regarded as a Mendelian character. It is well known that certain diseases are limited to one sex, though they are transmitted by both, such as hæmophilia, peroneal atrophy, colour-blindness, etc. Transmission to males through an unaffected female is called by Bateson the " knight's move " in heredity. Wood's experiments, in which horned Dorset were crossed with hornless Suffolk sheep, show that the presence of horns in these sheep is dominant in the male and recessive in the female. Probably those rare cases of colour-blindness in which females are affected as well as males belong to this group, for in all the instances recorded the affected female invariably transmits the disease to all her offspring, which is in accordance with what would be expected.

The establishment of the Mendelian nature of a disease demands that full and accurate pedigrees be obtained, and it is to be noted that the number and relationship of the normal individuals is quite as important as of the abnormal. When once the question is settled in the affirmative it is possible to predict always the probable, sometimes the inevitable result of a given mating. Thus, when a brachydactylous man marries a normal woman there is an even chance of any given child being diseased or normal, and when two normal people with night-blind parents and grandparents marry none of their children will inherit the disease (Punnett).

BATESON.—Brain, xxix, 1906. *PUNNETT.—Mendelism, 2nd edit., 1907; R. Soc. of Med., Epidemiological Section, 1908. *THOMSON.—Heredity, London, 1908.

It will be convenient to review briefly here the diseases of the eye in which the influence of heredity appears to be specially prominent. Some have already been discussed elsewhere and require no further consideration.

Lids.—*Distichiasis.*—Transmission of distichiasis has been recorded from father to daughter (Wood), from mother to two sons (Westhoff); collateral inheritance in three siblings is recorded by Koller.

WOOD.—Chicago Ophth. and Otol. Sec., 1898. WESTHOFF, KOLLER.—C. f. A., xxiii, 1899.

Ptosis and epicanthus.—Congenital ptosis has been observed in father and daughter (Rampoldi), mother and daughter (Zweig), in three generations (Münden). Steinheim reports ptosis and epicanthus in five generations, one member, either male or female, in each generation being affected; similarly Sattler a man and his four sons. Vignes records ptosis, epicanthus, and blepharophimosis in four generations.

Epicanthus alone was observed by Manz in five out of ten siblings. Ptosis is often present with other defects of ocular movements (*v.* p. 1414).

Manz.—In G.-S., ii, 1876. Rampoldi.—Ann. di Ott., xv, 1885. Vignes.—Rec. d'O., 1889. Sattler.—T. Am. O. S., 1897. Steinheim.—C. f. A., xxii, 1898. Münden.—Deutsche med. Woch., 1899. Zweig.—B. z. A., xli, 1899.

Cornea.—*Nodular Opacity* (*v.* Vol. I, p. 245) and *Reticular Opacity* (*v.* Vol. I, p. 247) are essentially familial diseases in which there is little evidence of direct inheritance.

Congenital Opacity (*v.* Vol. III, p. 785) and *Congenital Anterior Staphyloma* (*v.* Vol. III, pp. 786, 772) are doubtful examples of true hereditary influence ; they are more probably inflammatory in origin.

Iris.—*Persistent Pupillary Membrane.*—See Vol. III, p. 795.

Corectopia.—*See* Vol. III, p. 799.

Schwarz.—Schmidt's Jahrbuch, xxxvii, 1843. v. Graefe.—A. f. O., ii, 1, 1855. Macnaughton Jones.—Dublin Jl. of Med. Sc., 1879. Breitbarth.—Dissertation, Giessen, 1878. Pufahl.—C. f. A., iii, 1879. Frickhöffer.—Dissertation, Bonn, 1880. Wicherkiewicz.—A. f. O., xxxiv, 4, 1888. Best.—A. f. O., xl, 4, 1894 (Bibliography).
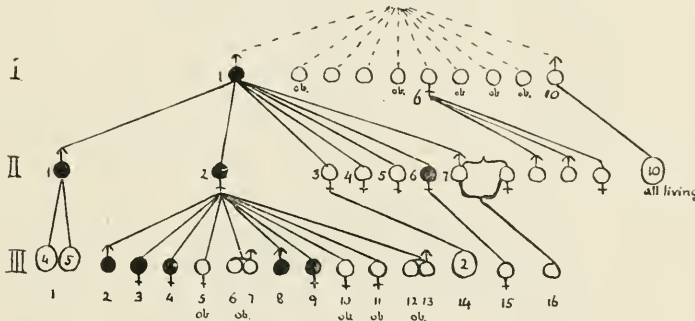
Fig. 857.—Lamellar Cataract.
Nettleship, R. L. O. H. Rep., xvi.

Polycoria.—*See* Vol. III, p. 801.

Aniridia.—*See* Vol. III, p. 802. The influence of heredity is seen in this anomaly more than in any other ocular malformation.

Gutbier.—v. Ammon's Z. f. O., v, 1834. Schröter.—K. M. f. A., iv, 1866. Page.—Lancet, 1874. Manz.—K. M. f. A., xiii, 1875. Laskiewicz-Friedensfeld.—K. M. f. A., xv, 1877. Benton, Rainsford —Brit. Med. Jl., 1878. Galezowski.—Rec. d'O., 1880. Pflüger.—Bericht d. Universitäts-Augenklinik, Bern, 1882. de Beck.—A. of O., xv, 1886 ; T. Am. O. S., 1894 ; A. of O., xxiii, 1894. de Benedetti.—Ann. di Ott., xv, 1886. Nicolini.—Boll. d'Oc., ix, 1887. Theobald.—Am. Jl. of O., 1888. Tokkus.—Dissertation, Strasburg, 1888. Strzeminski.—Gaz. lek., 1889. Mohr.—Dissertation, Jena, 1895. Carra.—Policlinico, 1899.

Lens.—*Congenital cataract.*—*See* Vol III, pp. 805, 1025. Hereditary influence is most strongly marked in the so-called *coralliform cataract* (*cf.* Fig. 567).

Lamellar cataract.—*See* Vol. III, p. 1025. Nettleship has published details of twenty-two families in which lamellar cataract was found in several generations. The most striking genealogy is shown in Fig. 857.

*Nettleship.—R. L. O. H. Rep., xvi, 3 and 4, 1905 (Bibliography).

Disciform post-nuclear cataract.—In 1905 I recorded a case of disciform opacity apparently situated in the posterior cortex of the lens imme-
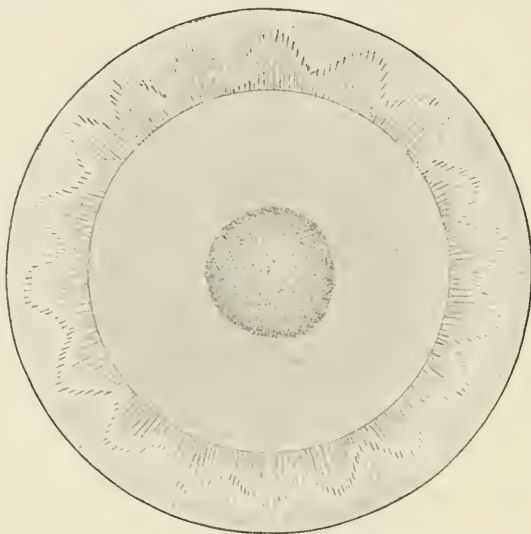
FIG. 858.—DISCIFORM POST-NUCLEAR CATARACT.
Nettleship and Ogilvie, T. O. S., xxvi.   Typical form.   Left eye of William Coppock,
æt. 55 (Gen. v, No. 9).
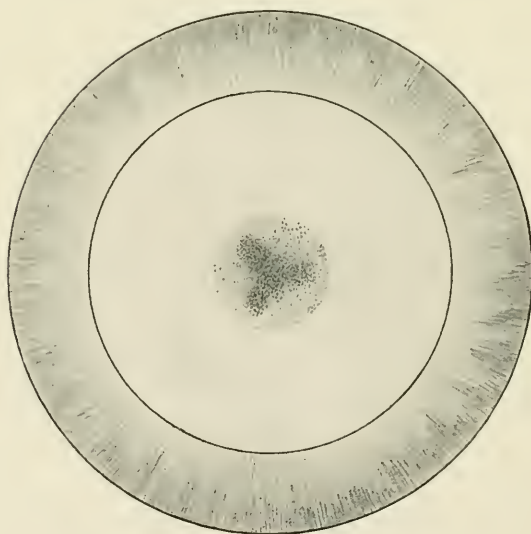
FIG. 859.—DISCIFORM POST-NUCLEAR CATARACT.
Nettleship and Ogilvie, T. O. S., xxvi.   Atypical form.   Left eye of Mrs. Packford,
æt. 24 (Gen. vi, No. 38).

diately behind the nucleus and affecting both eyes of a child (*cf.* Figs. 858—9).   In the following year Nettleship and Ogilvie published an

B. non - cataractous division
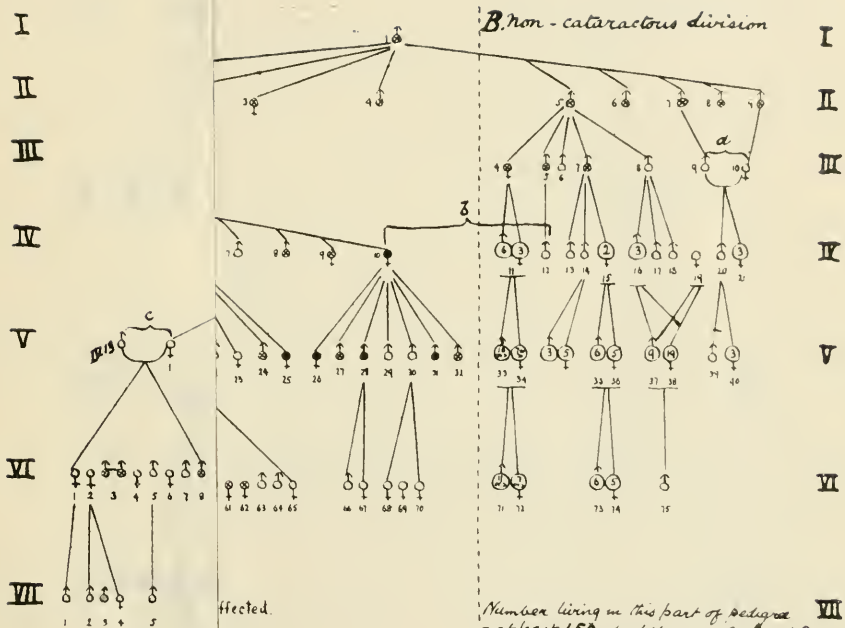
ffected.

affected

ormation

d by brackets (⌒) are cousins Four such are shown. There should be
of the sons of IV.16 and his 2nd cousin a daughter of one of IV.16. This
fathers and their 2 children are unaffected.

Number living in this part of pedigree = at least 150, doubtless more. Dead 8.
Of these, 48 (23 ♂ 25 ♀) had been examed.
up to June 1906 and all found normal.
including Nos 6, 1 and 9 ♀ Sea. III

A. Cataractous division   B. non-cataractous division

δ = Male   ♦ = Male affected
♀ = Female   ♦ = Female affected
○ = Dead, and no information

Husband & wife joined by brackets (⌐⌐) are cousins where such are shown. Here should be a fifth, between one of the sons of IV. 14 and his 27th cousin a daughter of one of IV. 16. This pair, their respective fathers and their 2 children are unaffected.

Total number on this part of pedigree   134.
                    Living .   101
                    Dead .   33
                    Examined .   90
                    affected . 18 normal . 72

Number living on this part of pedigree at least 150. somewhere most dead B. Of these, 68 (33 δ 25 ♀) had been examined. up to time (4·90) and all found normal.

FIG. 860.—DISCIFORM POST-NUCLEAR CATARACT
Nettleship and Ogilvie, T. O. S., xxvi.

extraordinary series of these cataracts in a family named Coppock (Fig.
860). Since then Chance has reported another family consisting of
father, mother, five sons, and one daughter, of whom the father, third,
fourth, and fifth sons, and the daughter were similarly affected.

PARSONS.—T. O. S., xxv, 1905. *NETTLESHIP AND OGILVIE.—T. O. S., xxvi, 1906.
CHANCE.—T. Am. O. S., 1907.
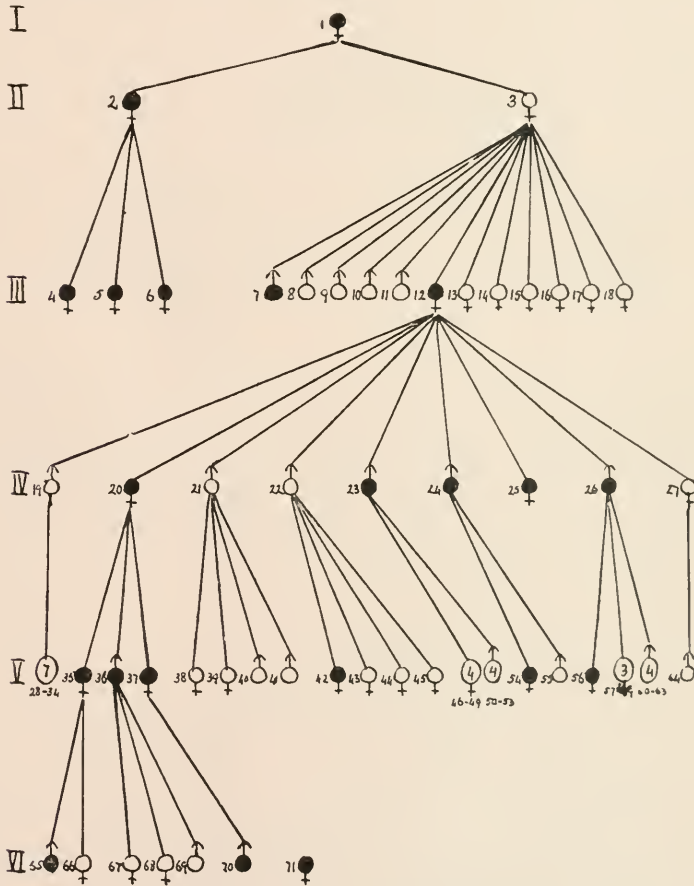
FIG. 861.—SENILE CATARACT.
Nettleship, R. L. O. H. Rep., xvi.   Green's case.

Other forms of congenital cataract.—Nettleship has collected a few
cases of other forms of congenital cataract which showed an hereditary
tendency.

NETTLESHIP.—R. L. O. H. Rep., xvi, 3 and 4, 1905.

Senile and pre-senile or juvenile cataract.—*See* Vo III, p. 1025.
Nettleship has collected a large number of cases of acquired cataract in
which hereditary influence has played a part.   His chief conclusions
have already been stated.   Five of the most striking genealogies of

senile cataract are shown in Figs. 861—865. The best example of
juvenile cataract is shown in Fig. 866.

*NETTLESHIP.—R. L. O. H. Rep., xvi, 3 and 4, 1905 (Bibliography).

Ectopia lentis.—*See* Vol. III, p. 809.
Retina.—*Retinitis pigmentosa.*—Nettleship has collected and analysed
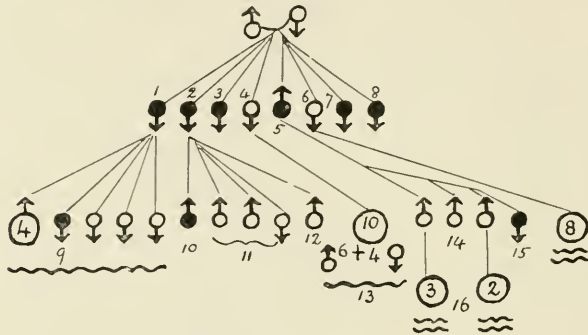
FIG. 862.—SENILE CATARACT.
Nettleship, R. L. O. H. Rep , xvi.　A wavy line means that the order of birth is unknown.

976 families, containing 1681 persons, with retinitis pigmentosa, and
over 50 families with allied diseases—retinitis punctata albescens
(Mooren and Gayet), 11 families with 20 affected persons ; atrophia
gyrata choroideæ et retinæ (Fuchs), 4 families with 10 persons ; con-
genital stationary night-blindness, 36 families with 260 members,
excluding Cunier's family (*v.* p. 1400).
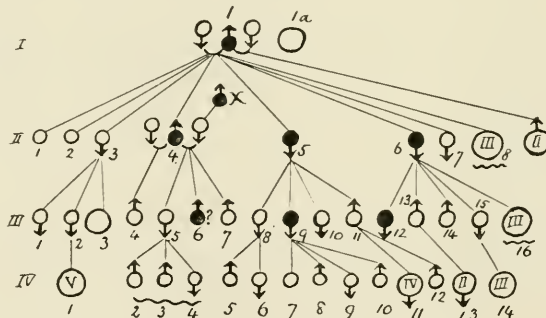
FIG. 863.—SENILE CATARACT.
Nettleship, R. L. O. H. Rep., xvi.

In the 976 families with retinitis pigmentosa with or without pigment,
there is evidence of heredity without consanguinity in 230 (23·5 per
cent.), of consanguinity without heredity in 226 (23 per cent.), and of
heredity combined with consanguinity in 32 (3—4 per cent). The most
extensive pedigrees are shown in Figs. 867—8. From a review of the
cases Nettleship concludes that it is certain that heredity is a potent
cause of retinitis pigmentosa, and that the descent, though sometimes

continuous from generation to generation, either directly from parent
to child or indirectly from uncle or aunt to nephew or niece, often
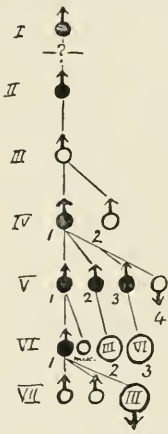
FIG. 864.—SENILE CATARACT.
Nettleship, R. L. O. H. Rep., xvi.
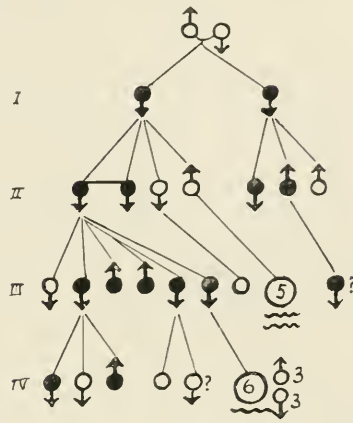
FIG. 865.—SENILE AND PRE-SENILE CATARACT.
Nettleship, R. L. O. H. Rep., xvi.

shows itself intermittently, and may lie hidden for as many as three or
even four generations. Of parental consanguinity more cannot at
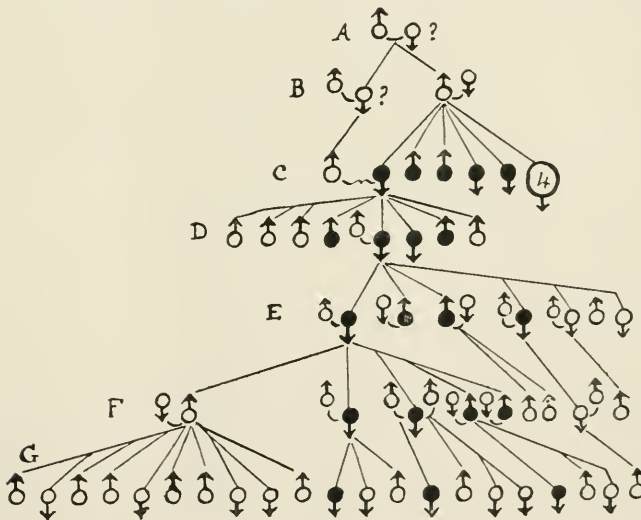
FIG. 866.—JUVENILE ACQUIRED CATARACT.
Nettleship, R. L. O. H. Rep., xvi.   Berry's case.

present be affirmed than that it often precedes the appearance of the
disease, and although its specific influence cannot be disproved, all the
indirect evidence points to marriage of blood relations being harmful
only when both husband and wife are members of a tainted stock.

Fig. 868.—Retinitis Pigmentosa.
Nettleship, R. L. O. H. Rep., xvii.
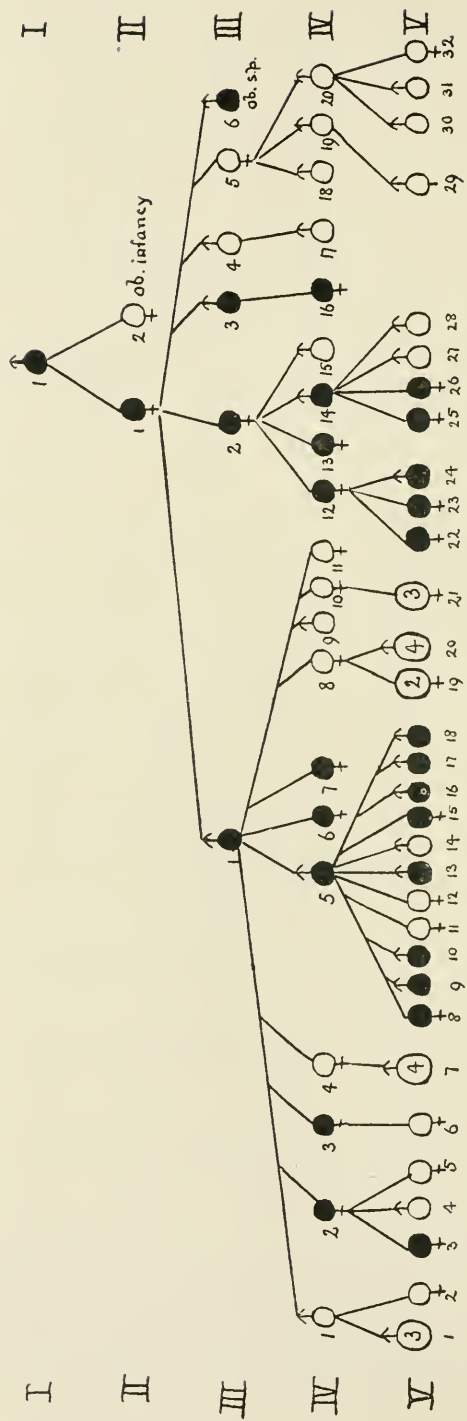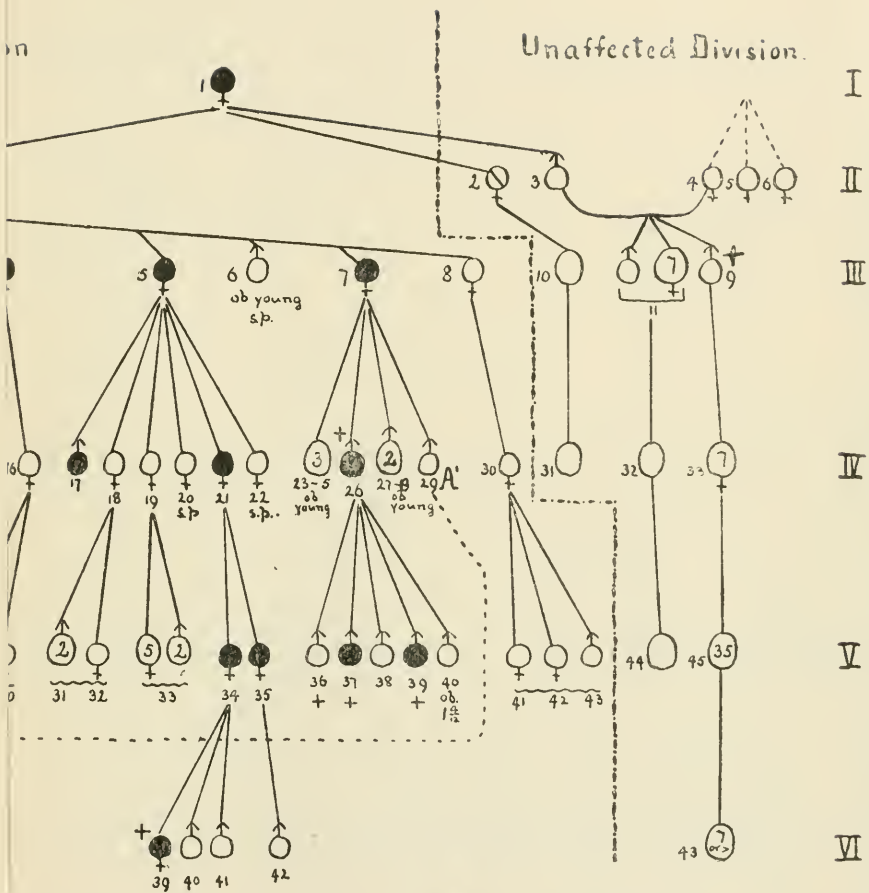
Total number of persons - - - - - - - - 73.($\male$ 39; $\female$ 34)

" " " with Retinitis Pigmentosa 29.($\male$ 13; $\female$ 16)

Unaffected Division.

I

II

III

Ⅳ

Ⅴ

Ⅵ

ⅦII

ob young
s.p.

ob young
s.p.

ob
young

23-5
ob
young

27-8
ob
young

A

44   45 35

ob.
1

43 or 3

have been seen and proved to have R.P. if ⬤, not to have R.P if ◯.
ersons in Affected Division, 200. (♂ 83 , ♀ 77; sex not stated 40)
"   with Retinitis Pigmentosa , 38 (♂ 20; ♀ 18)

FIG. 867.—RETINITIS PIGMENTOSA
Nettleship, R. L. O. H Rep., xvii.

Those marked + have been seen and proved to have R.P. if ●, not to have R.P if ○.
Total number of persons in Affected Division, 200. (♂ 83, ♀ 77; sex not stated 40)
"   "   "   "   " with Retinitis Pigmentosa, 38 (♂ 20; ♀ 18)
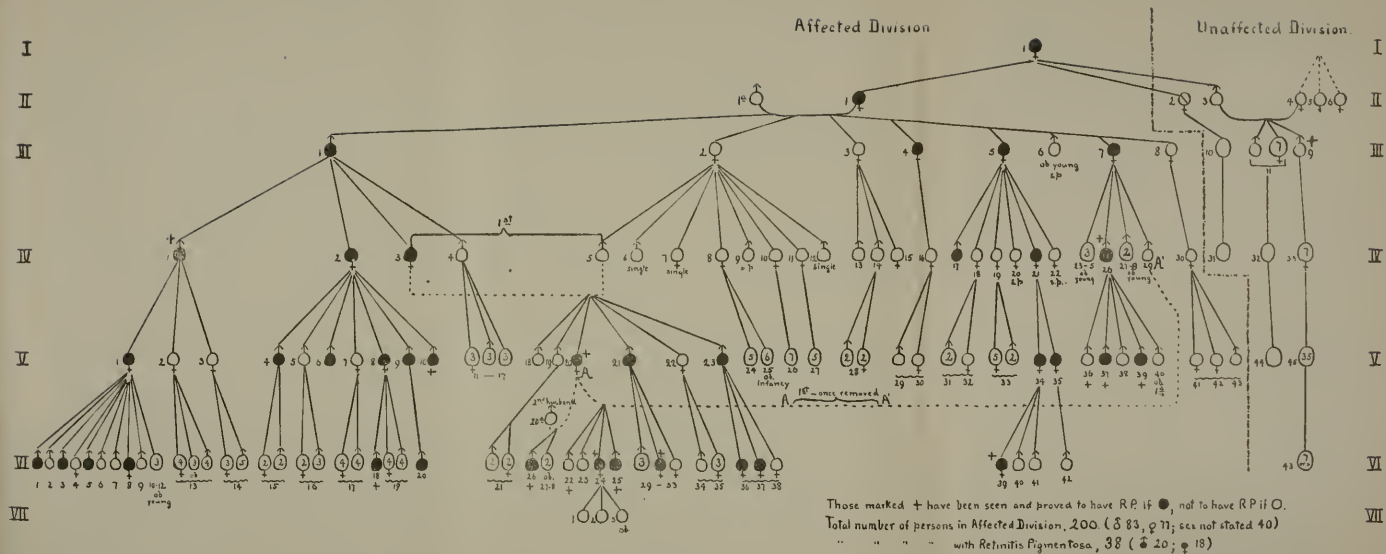
FIG. 869.—RETINITIS PIGMENTOSA.

Number of Affected and Not-Affected Children of each Sex in each Childship of (A) Not-Consanguineous Parentage: (B) Consanguineous Parentage. (Nettleship.)

All childships that were probably incomplete, all in which children died too early for state of sight to be known (except a few in which the proportion of such early deaths to the whole number born was insignificant), and all in which either parent was affected, are omitted.

Number of childships	Affected			Not affected			Total of both sexes		
	Male	Female	Total	Male	Female	Total	Affected	Not affected	
(A) Not Consanguineous.									
93	118 sex not stated	103	21 } 242	115 sex not stated	134	100 } 349	242	349 } 591	Average number of children to each childship . 6·3 / Max. 13. Min. 2. / Average number affected in each childship . 2·5
(B) Consanguineous.									
48	61 sex not stated	45	10 } 116	44 sex not stated	53	46 } 143	116	143 } 259	Average number of children to each childship . 5·5 / Max. 13. Min. 1. / Average number affected in each childship . 2·4

Thus the average number of children born of each parentage and the average number affected was almost identical for the consanguineous and not-consanguineous marriages.

The proportion affected was exactly or very near to half in each of 32 of the 93 not-consanguineous series, or about 33 per cent.

And the proportion affected was exactly or very near to half in each of 18 of the 48 consanguineous series, or about 37 per cent.

The extreme proportions of affected to healthy children in the separate childships were:—

(A) *Not-Consanguineous Series.*

1 affected in 9, 10, or 11 . . . . 5 times (minima).
8 ,, 11 . . . . . once } (maxima).
1 ,, 1 . . . . . once

(B) *Consanguineous Series.*

1 affected in 5 . . . . twice (minimum).
1 ,, 1 . . . . twice (maximum).

i.e., the minimum proportion affected was higher in the consanguineous series; lower and more frequent in the other.

As regards sex, of 1381 cases in which this detail is mentioned there were 845 males and 536 females, or about 3 to 2. Transmission by the father occurred in 36 families—to sons only 14, to daughters only 16, to both sons and daughters 3, to children, sex not stated 3. Transmission by the mother occurred in 50 families—to sons only 18, to daughters only 15, to both sons and daughters 15, to children, sex not stated, 2. In discontinuous transmission in 5 the disease passed from grand-parent through normal son to grandchild, in 3 through normal daughter. Hence it appears that though there are more living males than females suffering from retinitis pigmentosa, the disease is transmitted oftener by the affected women than by the affected men, probably because the malady acts as a greater bar to marriage in men than women. Further, the sex of the transmitting parent has no influence on the sex incidence of the disease in the children.

*Nettleship.—R. L. O. H. Rep., xvii, 1, 1907 (Bibliography).

Congenital night-blindness.—In this rare disease there is night-blindness without changes in the fundus, and with usually normal
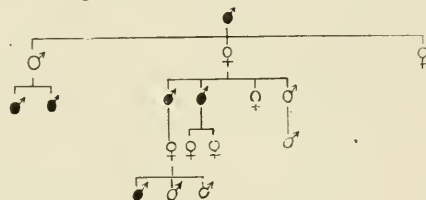
Fig. 871.—Congenital Night-blindness.
Pagenstecher's case.

visual acuity, and full fields with bright illumination. The first and most important family history is that recorded by Cunier (1838), and carried down to the present day by Nettleship (1907). The first known member of this family, himself night-blind, was Jean Nougaret, born in
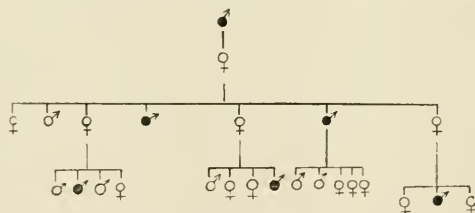
Fig. 872.—Congenital Night-blindness.
Cutler's case.

1637. The family inhabit Vendémian and the surrounding villages, near Montpellier. The pedigree, as completed by Nettleship and Truc, consists of 2121 persons, 1001 male, 960 female, and 160 whose sex is not known. Of the 2121, 135 are known to be, or have been, night-blind, 72 males, 62 females, and 1, sex not stated (Fig. 870). As is shown by the pedigree, the disease is very unequally distributed. The

37 to 1907.
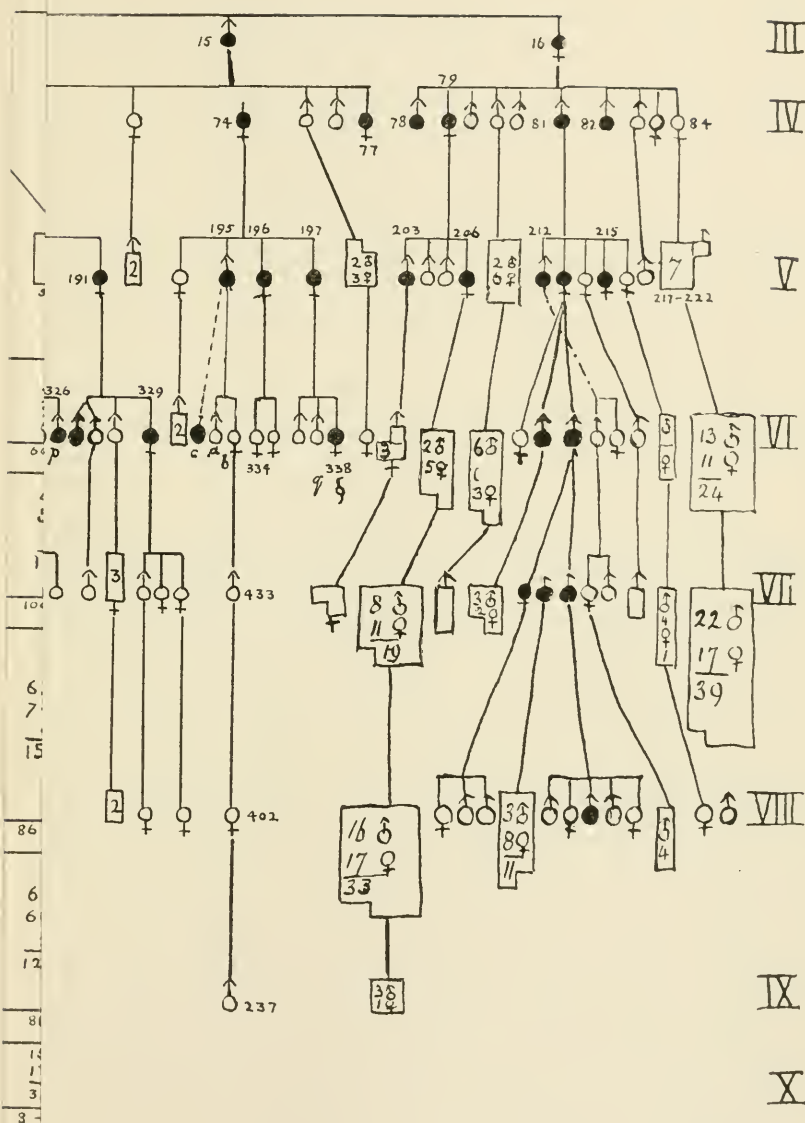
Jean Nougaret. Born 1637. Died 1719. Congenital stationary Night-blindness. The Nougaret family of Vendémian. 1637 to 1907.
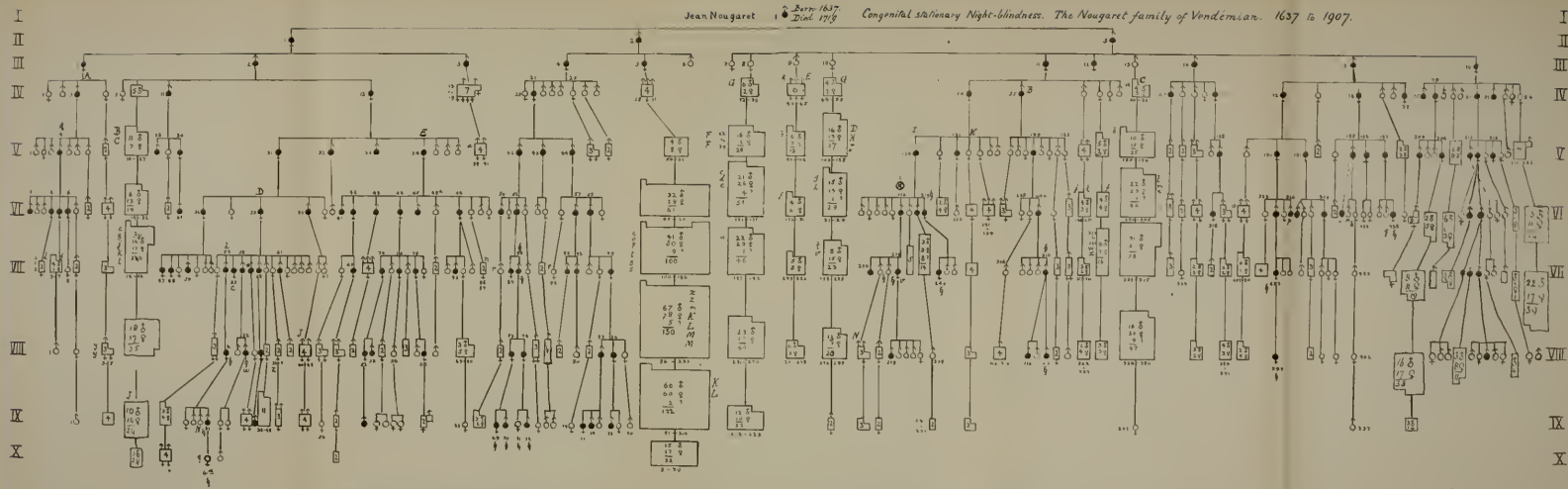
Signs:—Male ♂ normal ● night-blind
Female ♀ normal ● night-blind
⊕, 1st case (VI, 216) described by Cunier, 1838
I, examined by Professor Truc and Mr Nettleship, 15 of these night-blind, 2 normal, 1 (X, 4, too young, not test

Total of Pedigree 2116: male 1111, female 961, remainder no record of sex.
Total known night-blind 135, ♂ 83, ♀ 51, sex not recorded 1.
Each rectangular enclosure, drawn to scale, contains normal persons, as many as its contained numerals indicate.
Selected members of each generation numbered left to right.
Pairs of letters, a to z and A to N, husbands and wives: thus, q, VII, 90, is husband of q, VI, 138.

Fig. 870.
Nettleship. I. O. S. XXVII.

family tradition that the healthy child of a night-blind parent never carries the disease is fully borne out. There is no alternation of healthy with night-blind generations (discontinuous inheritance), no skipping of one or more generations, no reversion, no atavism in the generally accepted sense. The descent is invariably continuous. The disease-bearing branches consist of 255 persons, of whom 135 (53 per cent.) are affected. Of the 255, 139 (54·7 per cent.) are males, 115 (45·3 per cent.) females ; of these 72 males (52 per cent.) and 62 females (54 per cent.) are affected. Hence, again in accordance with the family
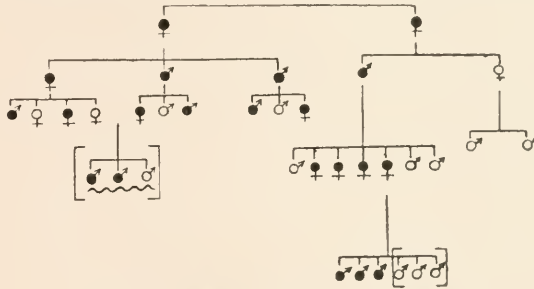
FIG. 873.—CONGENITAL NIGHT-BLINDNESS.
Sedan's case, with additional members (in brackets), from Cutler's paper.

tradition, the females appear to be rather more liable than the males. It will be seen that the affected men married less freely than the affected women, and of those who did marry the proportion who had night-blind offspring was decidedly smaller than in the case of the women.

As regards consanguinity, there are 40 consanguineous and 717 doubtful or non-consanguineous marriages. In the total number of
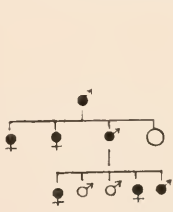
FIG. 874.—CONGENITAL
NIGHT-BLINDNESS.
Atwool's case.
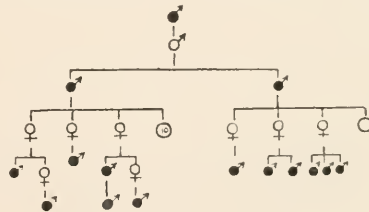
FIG. 875.—CONGENITAL NIGHT-
BLINDNESS.
Morton's case.

childships (71) containing one or more affected children, only 8 are known to have been the issue of cousins, and in all but one the relationship was less than that of second cousins. Evidently cousinship, as such, does not produce the disease.

Other observers have reported families affected with congenital night-blindness. Pagenstecher's pedigree includes five generations, of which the second and fourth were free, an example of discontinuous inheritance which is absent from the Cunier-Nettleship family. Only males were affected. The pedigree is shown in Fig. 871. Cutler's pedigree also shows discontinuous inheritance (Fig. 872) ; there were no

consanguineous marriages. In no case was the firstborn child affected. In Sedan's pedigree inheritance is always continuous (Fig. 873), as also in Atwool's (Fig. 874). Morton's pedigree, again, shows discontinuous inheritance (Fig. 875).

In Ammann's pedigree transmission is invariably by "knight's move" to sons only, *i. e.* through unaffected females. In the four generations the number of affected persons gradually diminishes. Collateral heredity is shown in Swanzy's family, in which 3 sisters and 2 brothers were affected out of 5 brothers and 5 sisters, and in Förster's with 2 brothers affected. In only two reported examples does consanguinity of parents appear (Leber, Donders).

In Cutler's collation of 10 pedigrees, which exclude the Cunier-Nettleship case, there are 54 affected persons, 36 male and 18 female. In 3 of the families men alone were affected. Comparing these results, which are emphasised by later examples, such as that of Ammann, with the Cunier-Nettleship case, it will be evident that general deductions on sex transmission, etc., are liable to be fallacious.

CUNIER.—Ann. de la Soc. de Méd. de Gand, 1838; Ann. d'Oc., i, 1838. STIEVENART.—Ann. d'Oc., xviii, 1847. DONDERS.—Nederl. Lancet, 1854. FOERSTER.—Ueber Hemeralopie, Breslau, 1857. MAES.—Jahresbericht d. Utrechter Augenklinik, 1861. SWANZY, FITZGERALD.—Irish Hosp. Gaz., 1873. LEBER.—In G.-S., v, 1877. PAGENSTECHER.—C. f. A., ii, 1878, Beilageheft. VIEUSSE.—Gaz. hebd., 1878. PFLÜGER.—Jahresbericht a. d. Universitäts-Augenklinik in Bern, 1881. ZIMMERMANN.—A. of O., xii, 1883. CHIBRET.—A. d'O., iv, 1884. SEDAN.—Rec. d'O., 1885. NETTLESHIP.—R. L. O. H. Rep., xi, 1887. MORTON.—T. O. S., xiii, 1893. CUTLER.—A. of O., xxiv, 1895. ATWOOL.—R. L. O. H. Rep., xiv, 1895. AMMANN.—Correspondenzbl. f. Schweizer Aerzte, 1898. NETTLESHIP.—T. O. S., xxvii, 1907.

Optic nerve.—*Hereditary optic neuritis* (Leber).—This disease was first described by Leber in 1871. The earliest recorded case is probably that of v. Graefe (1858); all cases previously published were collected by Klopfer (1898) and Hormuth (1900).

In almost all cases (70 out of 74, Hormuth) the disease manifests itself as a retrobulbar neuritis, usually at about the twentieth year of life. Vision generally fails rapidly at first, then gradually, then remains stationary or gradually improves. Occasionally failure is rapid and may cause complete blindness, but may again improve (Leber). Both eyes are always affected, though one may precede the other by a few days up to eighteen months. In other cases failure of vision may be very slow, extending over a year. In two thirds of the cases there is a central scotoma, either partial for colours or also for white. The peripheral field is usually normal, but concentric contraction for white and colours, or sector-shaped defects may occur. Total and permanent colour-blindness has been known to ensue. The central scotoma generally persists, though improvement of central vision, even up to normal, may occur. Progressive constriction of the field to complete blindness is rare. Members of the same family often show identical peculiarities in the progress of the cases.

The fundus is at first normal, or there is slight blurring of the edges of the disc, the arteries being normal or even widened. In later stages, after months, optic atrophy ensues, with pallor confined to the temporal side or involving the whole disc. Hotz has published the

record of a family in which apparently progressive optic atrophy occurred without the signs of preceding neuritis. Two sisters and a brother were affected at the ages of fifteen, twenty-two, and eighteen respectively. Headaches, migraine, etc., are frequently recorded, but serious general disease is rare. Occasionally other members of the family have suffered from retinitis pigmentosa, or colour-blindness.

The cause of the disease is unknown. Syphilis is reported in only two cases (Higgins, Linde). Berger attributes the condition to anomaly in the development of the sphenoid; but cases to which such an explanation is applicable belong to another group (Müller, Vossius). Velhagen has recourse to the greater vulnerability of the papillo-macular fibres of the optic nerve, but does not explain the mode in which they are attacked.

Klopfer's statistics include 48 families with 214 affected persons;
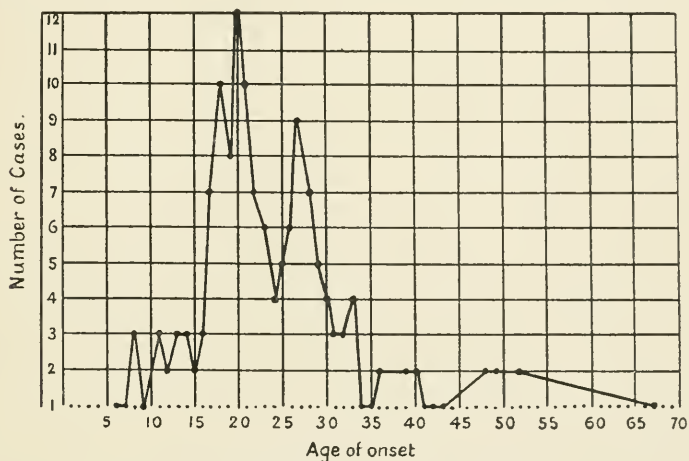
Fig. 876.—Hereditary Optic Neuritis.
Hancock, R. L. O. H. Rep., xvii.

Hormuth's 65 families with 284 cases. The age of onset is usually 18 to 23 (Fig. 876), but varies from 5 to 67. In a smaller group, consisting mostly of women, the onset was from 41 to 49 (Habershon, Hormuth), so that in the rare cases in which women are affected the cessation of the menses appears to be an exciting cause. Klopfer in 214 cases found the usual age of onset to be 24, or, if a few doubtful cases are eliminated, 20. Habershon in 50 cases found the age between 13 and 32, i. e. about puberty. The younger siblings are likely to be affected at an earlier period of life than the older in any given childship, and a similar rule applies to later generations.

As regards sex, Hormuth in 298 cases found 262 men (88 per cent.) and 36 women (12 per cent.); in the 74 families in 72 per cent. only men were affected, in 28 per cent. both men and women. There are no families in which only women were affected. In only 2 cases were more women than men affected (Holz, Batten).

Transmission is almost always through an unaffected mother to the

sons. Direct descent occurs in only 14 per cent. of families; in 48 families twice through the father and 5 times through the mother. In

FIG. 877.—HEREDITARY OPTIC NEURITIS.
Hancock, R. L. O. H. Rep., xvii.

2 of these families transmission through the affected father or mother runs through several generations (Norris, Rampoldi). Collateral inheritance occurs in 32 of 71 families. Children of the same mother, but of two different fathers, may be affected (Westhoff).

Consanguinity of ancestors occurs only 3 times in 48 families (Mooren, König, Klopfer).

v. Graefe.—A. f. O., iv, 2, 1858; K. M. f. A., iii, 1865. Sedgwick.—Med. Times and Gaz., 1862. Mooren.—Ophthalmiatrische Beobachtungen, Berlin, 1867; Ophth. Mitt., Berlin, 1873. *Leber.—A. f. O., xvii, 2, 1871; in G.-S., v, 1877. Hutchinson.—R. L. O. H. Rep., vii, 1871. Daguenet and Galezowski.—Jl. d'O., i, 1872. Prouff.—Thèse, Paris, 1872. Alexander.—K. M. f. A., xii, 1874. Pufahl.—Berliner klin. Woch., 1876; Hirschberg's Beiträge, iii, 1878. Fuchs.—K. M. f. A., xvii, 1879. Higgins.—Med. Times and Gaz., 1879. Norris.—T. Am. O. S., 1882, 1885. Schlüter.—Dissertation, Bonn, 1882. Rampoldi.—Ann. di Ott., xii, 1883. de Keersnaecker.—Rec. d'O., 1883. Story.—Trans. Acad. of Med. of Ireland, iii, 1885. Holz.—Dissertation, Griefswald, 1885. Gevers.—Dissertation, Berlin, 1887. Browne, Habershon.—T. O. S., viii, 1888. Harwell.—Brit. Med. Jl., 1888. Thomson.—Deutsche med. Woch., 1888. Nicolai.—Weekbl. v. h. Nederl. Tijdsch., i, 1888. Taylor.—T. O. S., x, 1890. Sym.—Edin. Med. Jl., 1891. Despagnet.—Rec. d'O., 1892. Somya.—K. M. f. A., xxx, 1892. Thompson.—T. O. S., xii, 1892. Gould.—Am. Jl. of O., 1893. Müller.—K. M. f. A., xxxi, 1893. König.—Soc. franç. d'O., 1894; Ann. d'Oc., cxi, 1894. Linde, Westhoff.—C. f. A., xix, 1895. Dodd.—Ann. of Ophth. and Otol., 1895. Ogilvie, Batten.—T. O. S., xvi, 1896. Kauffmann.—Lancet, 1896. Velhagen.—Deutsche med. Woch., 1896. Snell.—T. O. S., xvii, 1897. Higier.—Deutsche z. f. Nervenheilk., x, 1897. Leitner.—Szenezet, 1897; Ungar, B. z. A., 1899. Lor.—Cercle méd. de Bruxelles, 1897. *Klopfer.—Dissertation, Tübingen, 1898. Posey.—Ann. of O., 1898. Brisson.—Thèse, Paris, 1899. Magers.—Dissertation, Jena, 1899. Strzeminski.—Ann. d'Oc., cxxi, 1899. *Hormuth.—B. z. A., xlii, 1900. Vossius.—Sammlung, iii, 1900. Asmus.—Die ophth. Klinik, 1901. Stood.—K. M. f. A., xxxix, 1901. Nettleship.—T. O. S., xxiii, 1903. Bickerton.—T. O. S., xxiv, 1904. Gunn, Lawson.—T. O. S., xxvi, 1906. Hancock.—R. L. O. H. Rep., xvii, 2, 1908.

Congenital optic atrophy.—There are cases of collateral inheritance of congenital optic atrophy on record, notably those of Jakobsohn—a boy and girl, fourth and seventh children, Newman 2 children, Snell 2 sisters and 3 brothers in a family of 8. Magnus in 113 cases found collateral inheritance in 14 of 2 or more siblings.

Magnus.—Jugendblindheit. Newman.—R. L. O. H. Rep., iv, 1864. Jakobsohn.—C. f. A., xi, 1887. Snell.—T. O. S., xvi, 1896.

Coloboma of the optic nerve.—Weyert saw coloboma of the optic nerve transmitted through 3 generations (Fig. 878). The grandfather had the coloboma in one eye, his two daughters in both, his grandchildren in one or both. One grandchild had coloboma of the iris.

Fig. 878.—Coloboma of the Optic Nerve.

Weyert's case. A had coloboma of the iris.

Weyert.—K. M. f. A., xxviii, 1890.

Lacrymal apparatus.—Emmert found absence of all four puncta in a boy, lacrymal fistulæ in his brother, and narrowing of the nasal duct in another brother; the grandfather had epiphora. Nieden's cases are not truly hereditary except, perhaps, of a tuberculous diathesis. Peters records cases of hereditary congenital atresia of the nasal duct, due to delayed canalisation.

Emmert.—A. f. A., v, 1876. Nieden.—C. f. A., vii, 1883; A. f. A., xvi, 1886. Peters.—K. M. f. A., xxx, 1892.

Glaucoma.—Benedict (1842) first published any reference to hereditary influence in glaucoma, two sisters being affected, their

father and brother being subjects of gout. He thought that the
glaucoma was a manifestation of hereditary gout. v. Graefe (1869)

FIG. 879.—GLAUCOMA.
Lawford, R. L. O. H. Rep., xvii.
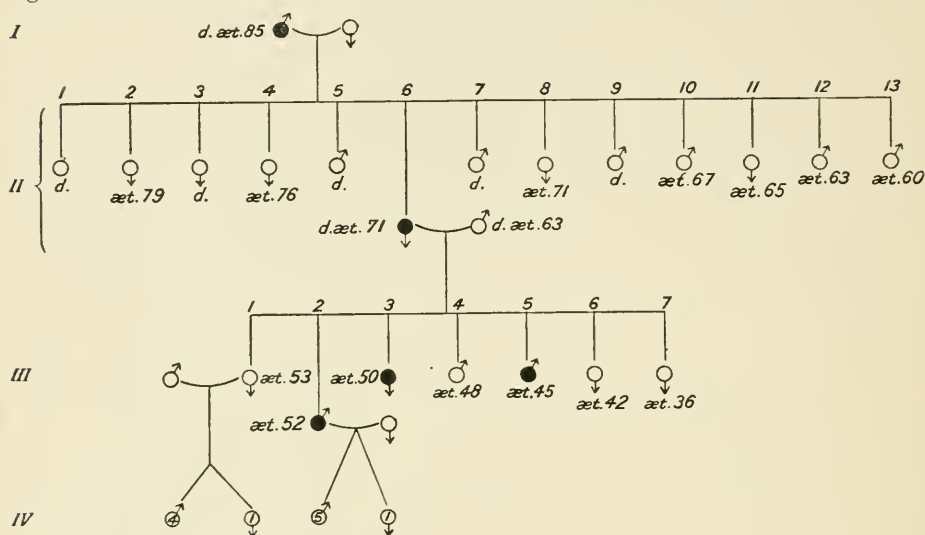
emphasised the importance of inheritance in the causation of glaucoma.
He says that "the influence of inheritance seems to predominate in

FIG. 880.—GLAUCOMA.
Lawford, R. L. O. H. Rep., xvii.
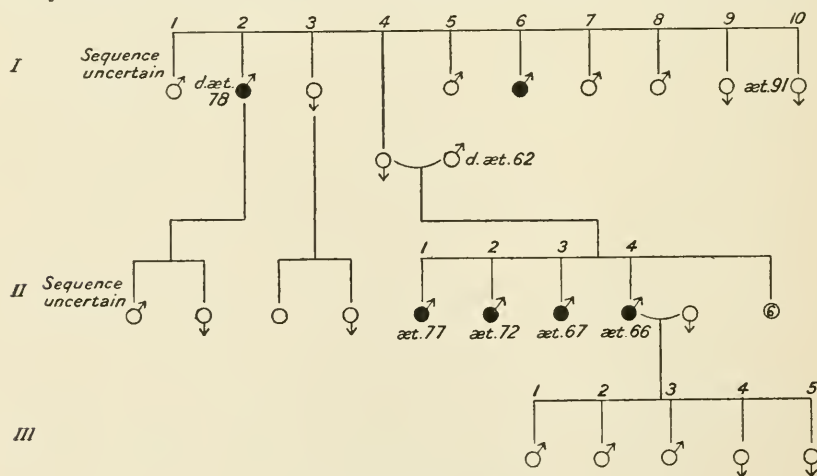
typical inflammatory glaucoma, which often attacks several members
of one family, and is transmitted from generation to generation. The
simple form has appeared to me far less frequently hereditary, but my

data are too scanty for any decision on this point." The records since
v. Graefe's time seem to show that hereditary glaucoma is more likely
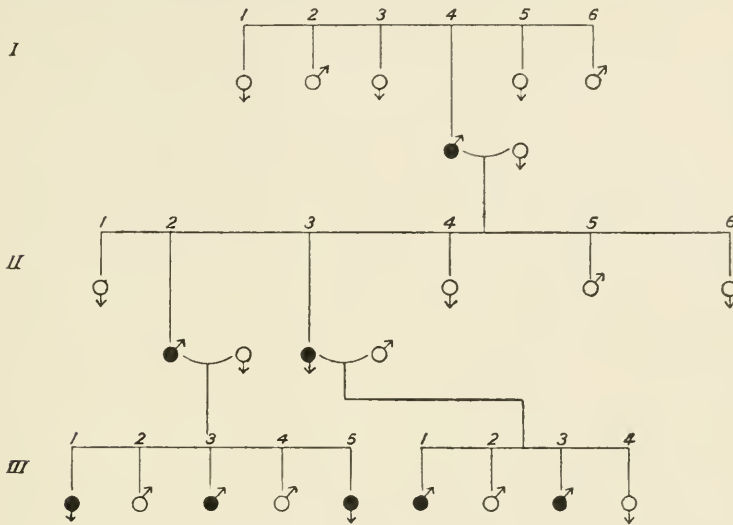
FIG. 881.—GLAUCOMA.
Lawford, R. L. O. H. Rep., xvii.   Howe's case.
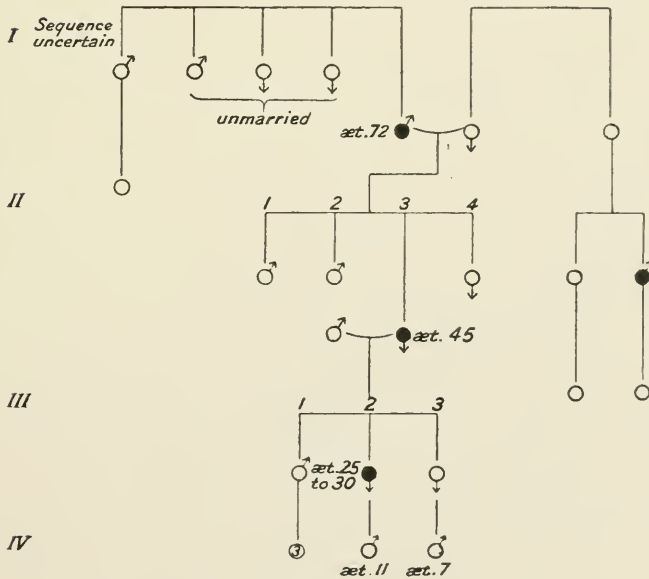
FIG. 882.—GLAUCOMA.
Lawford, R. L. O. H. Rep., xvii.   Nettleship's case.

to assume the chronic than the acute form (Lawford). v. Graefe
recognised the tendency to " anticipation " in hereditary glaucoma, *i.e.*

the tendency for the disease to appear at an earlier age in successive generations. He refers to families in which glaucoma affected three or four generations. "The first symptoms now usually appear at thirty
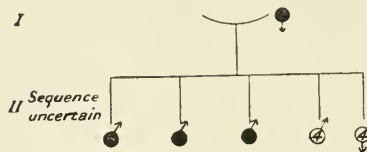
Fig. 883.—Glaucoma.
Lawford, R. L. O. H. Rep., xvii.
Rogman's case.
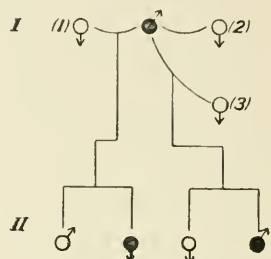
Fig. 884.—Glaucoma.
Lawford, R. L. O. H. Rep., xvii.
Somya's case.

or forty, whilst the parents and grandparents became affected at fifty or sixty." Anticipation is shown in the cases published by Mules, Somya, Müller-Kannberg, Rogmann, and Lawford. He also noticed in the inherited form an unusually long prodromal stage extending
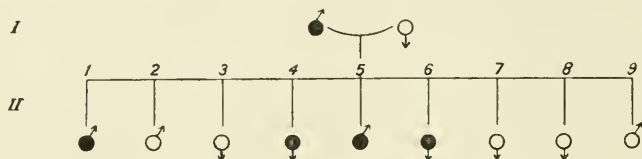
Fig. 885.—Glaucoma.
Lawford, R. L. O. H. Rep., xvii. Nettleship's case.

over ten or fifteen years, but this is characteristic of glaucoma in relatively young patients (Laqueur).

Direct inheritance is common, about half the cases being through the father and half through the mother. There seems to be no special
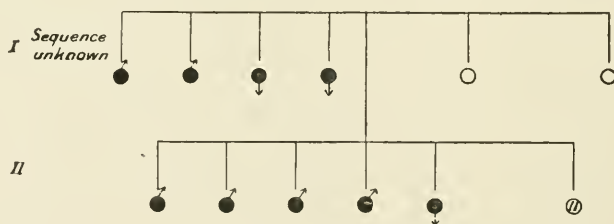
Fig. 886.—Glaucoma.
Lawford, R. L. O. H. Rep., xvii. Kummer's case.

tendency to transmission to the same sex, but males are slightly more liable to inherit the disease.

Collateral inheritance is exemplified in the cases of Mooren, 2 siblings; Pflüger, 2 brothers; Nolte, 2 brothers, and other cases. The

frequency of glaucoma in Jews is possibly due in some degree to inter-marriage. Wagner in 19,525 eye-patients found glaucoma in 1·61 per cent. of Christians, and 2·57 per cent. of Jews.

BENEDICT.—Abhandlungen aus dem Gebiete der Augenheilkunde, Breslau, 1842. v. ARLT.—Die Krankheiten des Auges, ii, Prag, 1853. STELLWAG.—Die Ophthalmologie, etc., ii, 1855. PAGENSTECHER.—Klin. Beobachtungen, 1861. MOOREN.—Ophthalmiatrische Beobachtungen, 1867. v. GRAEFE.—A. f. O., xv, 3, 1869. KUMMER.—Correspondenzbl. f. Schweizer Aerzte, 1871. PFLÜGER.—K. M. f. A., xiii, 1875. SCHMIDT-RIMPLER.—In G.-S., v, 1877. LAQUEUR.—A. f. O., xxvi, 2, 1880; xlvii, 1899. SCHENKL.—Prager med. Woch., 1880. MULES.—Ophth. Rev., 1883. WAGNER.—A. f. O., xxix, 2, 1883. MOOREN.—A. f. A., xiii, 1884. RAMPOLDI.—Ann. di Ott., xiii, 1884. HARLAN.—Jl. of Am. Med. Assoc., 1885, 1899. JACOBSON.—A. f. O., xxxii, 3, 1886. HOWE.—A. of O., xvi, 1887. SCHWEIGGER.—A. f. A., xxiii, 1891. SOMYA.—K. M. f. A., xxxi, 1893. MÜLLER-KANNBERG.—K. M. f. A., xxxii, 1894. PRIESTLEY SMITH.—Ophth. Rev., 1894. RUDIN.—In Nagel's Jahresbericht, 1895. NOLTE.—Dissertation, Marburg, 1896. LANGE.—Vossius' Sammlung, i, 1896. ROGMANN.—La Clinique ophth., 1899. NETTLESHIP.—R. L. O. H. Rep., xii, 1888; Ophthalmoscope, 1906. *LAWFORD.—R. L. O. H. Rep., xvii, 2, 1908.

Buphthalmia.—Under this designation, which probably includes not only infantile glaucoma, but also cases of keratoglobus which are possibly of different pathogenesis, cases of collateral inheritance are recorded: Manz, 3 brothers; Jüngken, 7 brothers; Streatfeild, 2 sisters; Warloment, 2 brothers. Laqueur found consanguinity of parents in four cases.

MANZ.—In G.-S., ii, 1876. STREATFEILD.—Lancet, 1882. WARLOMENT.—Ann. de la Soc. de Bruxelles, 1896.

Microphthalmia and anophthalmia.—Microphthalmia, of which anophthalmia is clinically only the extreme expression, often occurs in several children of the same parents. Manz records four examples, Bruns 3 out of 7 siblings. Lafosse reports transmission from mother to child, Landesberg from father to firstborn, Mayerhausen through three generations.

MANZ.—In G.-S., ii, 1876. LANDESBERG.—K. M. f. A., xv, 1877. MAYERHAUSEN.—C. f. A., vi, 1882. LAFOSSE.—A. of O., xxv, 1896. BRUNS.—Am. Jl. of O., 1899.

Ametropia.—The frequency of errors of refraction makes it a difficult problem to estimate the influence if any of heredity. Myopia in one or both parents of myopes has been found in the following percentages by various observers: Knies 21 per cent.; Erismann 31 per cent.; v. Hippel 50 per cent.; Kirchner 50 per cent.; Straumann 56 per cent.; Ohlemann 57 per cent.; Schmidt-Rimpler 26 per cent. in lower class schools, 49—76 per cent. in upper class schools; Galezowski 83 per cent.; Magnus 28·5 per cent. to 42·8 per cent. (Groenouw).

Pflüger has compared families with and without inherited myopia. In 100 families in lower schools, the parents not being myopic, 74 had no myopia; the total number of children was 449, of whom 8·4 per cent. had myopia, 91·6 per cent. other conditions of refraction. In 100 families of the same schools, one or both parents being myopic, 55 had no myopia; the total number of children was 395, of whom 19 per cent. had myopia, 81 per cent. other conditions of refraction. In higher schools, in 85 families, the parents not being myopic, 40 per cent. had myopic children; of the 280 children 17 per cent. were myopic. In 55 families of the same schools, one or both parents being myopic,

71 per cent. had myopia; of the 228 children 26 per cent. were myopic. Only rarely were all the children of a family myopic. Analysis of these statistics leads to the conclusion that only 10 per cent. show hereditary influence, which is too small a number to be decisive considering the numerous factors which are not taken into account.

Schneller, in 1439 scholars of higher schools in Danzig, found that 9 per cent. hypermetropes, 12 per cent. emmetropes, and 37 per cent. myopes had myopic parents. Kirchner found in 1156 children of 356 families, with one or both parents myopic, 31 per cent. of myopes, whilst in 2069 children of 630 families in which both parents were emmetropic or hypermetropic, only 15 per cent. were myopes.

Tscherning, in Danish conscripts, found 30 per cent. of myopes showed hereditary tendency—myopia of 2 D, 15 per cent.; 3—6 D, 33 per cent.; 7—9 D, 46 per cent.; and more than 9 D, 38 per cent.
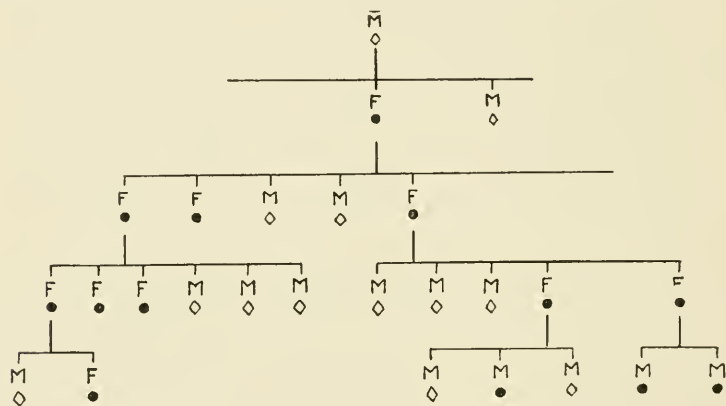
Ο myopic.   ● not known to be short sighted.

FIG. 887.—MYOPIA.
Worth, T. O. S., xxvi.

with myopic parents. Careful analysis of these results by the author in the original paper very considerably modifies their significance.

Extensive statistics of this nature, though lacking in precision, are of greater importance than sporadic examples of myopic families, such as those recorded by Theobald, Maclehose, Worth (Fig. 887), and others.

Considering the insecurity of the fundamental facts the question of sex transmission can scarcely be discussed with much advantage. Motais concluded from his statistics, 65 per cent. of cases of myopia having hereditary tendency, that transmission from the father was generally to the daughters, and from the mother to the sons. Magnus found that the father transmitted the affection to the sons in 30·6 per cent., to the daughters in 19·8 per cent.; the mother to the sons in 17·8 per cent., to the daughters in 14·8 per cent.; both parents to the sons in 4·5 per cent., to the daughters in 4·3 per cent. Schlesinger, in myopia of more than 6 D, found that inheritance from the father

occurred in 54·9 per cent.; from the mother in 25·6 per cent.; from both in 19·5 per cent. The father transmitted it to the sons in 28·6 per cent., to the daughters in 26·3 per cent.; the mother to the sons in 14·3 per cent., to the daughters in 11·3 per cent.; both to the sons in 14·3 per cent., to the daughters in 5·2 per cent.

Erismann found that the influence of the father was greater than that of the mother in both boys and girls. Leininberg found the father affected in 113 cases and the mother in 63 cases examined. These statistics are vitiated by the facts as to sex incidence of the disease (v. Vol. III, p. 922).

Consanguinity of parents appears to have little influence, though the opposite proposition is held by Laqueur and his pupil Wolff.

ERISMANN.—A. f. O., xvii, 1, 1871. PFLÜGER.—A. f. O., xxii, 4, 1876. EMMERT.—Auge u. Schädel, Berlin, 1880. TSCHERNING.—A. f. O., xxix, 1, 1883. SCHNELLER, KNIES.—A. f. O., xxxii, 3, 1886. LEININBERG.—Dissertation, Würzburg, 1886. GALEZOWSKI.—Ann. d'Oc., xcvii, 1887. STRAUMANN.—Dissertation, Basel, 1888. KIRCHNER.—Z. f. Hygiene, vii, 1889. MOTAIS.—A. d'O., viii, 1889. SCHMIDT-RIMPLER.—A. f. O., xxxi, 4, 1885; xxxv, 4, 1889. LEHMANN.—Dissertation, Kiel, 1890. JAVAL.—France méd., 1891. PROSKAUER.—A. f. O., xxxvii, 2, 1891. THEOBALD.—Johns Hopkins Hosp. Rep., 1891 ; Am. Jl. of O., 1891. COHN.—Hygiene des Auges, Wien, 1892. OHLEMANN.—A. f. A., xxvi, 1893. MAGNUS.—Bericht d. Augenanstalt in Breslau, 1895. WOLFF.—A. f. A., xxxiii, 1896. MACLEHOSE.—Ophth. Rev., 1897. SCHLESINGER.—B. z. A., xlv, 1900. WORTH.—T. O. S., xxvi, 1906.

Albinism.—Albinism appears to be scarcely ever transmitted by direct inheritance, though collateral inheritance and consanguinity of parents are found. Albinism is common in Sicily: Arcoleo, amongst the 254,000 inhabitants, found 62 albinos belonging to 24 families. Albinos seldom marry and are generally sterile ; marriage between two albinos was not seen. Six married albinos had no albinotic child. In 5 of the 24 families consanguinity of parents of the second degree occurred, and of the 43 children 14 were albinos. Consanguineous marriage of ancestors was found in the case of other albinos.

Collateral inheritance has often been noted—Streatfeild, three out of six children, Mayerhausen, and others. Alternate pigmented and albinotic children were born in the families reported by Sym and Abadie. In twins one may be quite normal, the other an albino (Arcoleo, Hutchinson). Albinism in two lines occurs (v. Förster, Koren). In v. Forster's case two boys and a girl were albinos, and some of the cousins by the maternal aunt showed the same anomaly. In Koren's case alternate albinos were born, and the mother's sister-in-law had an albino child.

The heredity of albinism is at present being investigated by Karl Pearson, Stainer, and Nettleship.

ARCOLEO.— Gaz. clin. dello Spedale civico di Palermo, ii, 1871. HUTCHINSON.—R. L. O. H. Rep., vii, 1871. KOREN.—Norsk. Magaz. f. Lägevid, 1877. MANZ.—A. f. O., xxiv, 4, 1878. ABADIE.—L'Union méd., 1879. v. FORSTER.—K. M. f. A., xix, 1881. MAYERHAUSEN.—K. M. f. A., xx, 1882. STREATFEILD.—Lancet, 1882. SYM.—Ophth. Rev., 1891.

Colour-blindness.—Colour-blindness occurs in about 3·69 per cent. of males and 0·088 per cent. of females (Norris and Oliver) ; probably the estimates for females are too low (Nettleship). Transmission

through several branches of a family is not rare, and several siblings, usually of course male, of a childship are often affected. Collateral
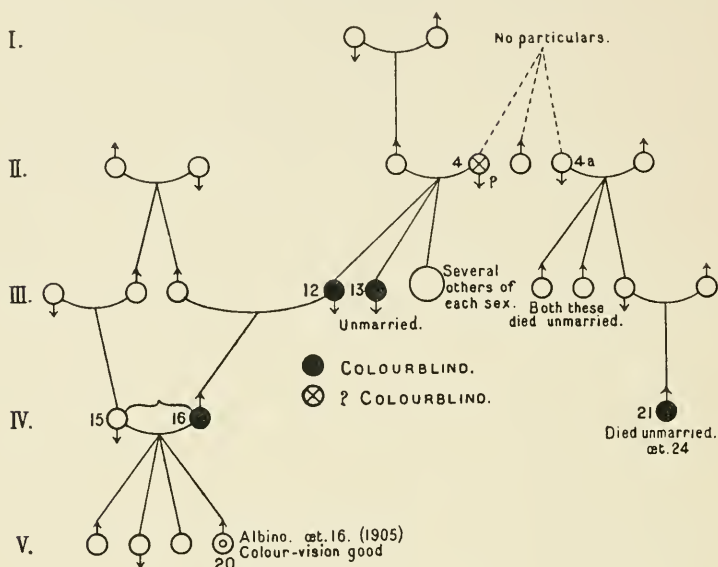
Fig. 888.—Colour-blindness.
Nettleship, T. O. S., xxvi.

inheritance of total colour-blindness is also recorded (Raehlmann, Pflüger). Horner first pointed out the rule that inheritance is usually through unaffected females, *i. e.* discontinuous inheritance by " knight's
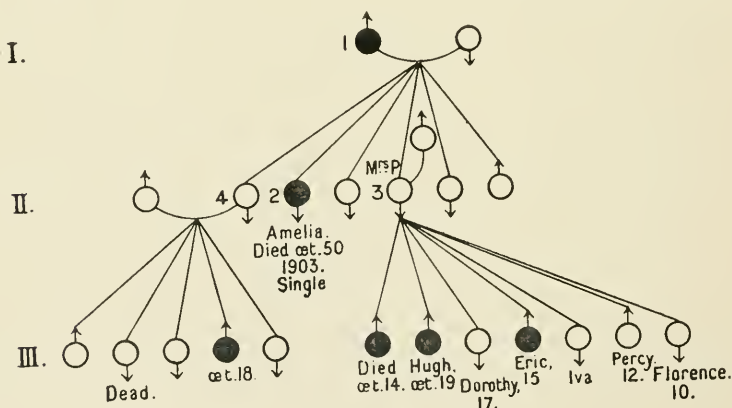
Fig. 889—Colour-blindness.
Nettleship, T. O. S., xxvi.

move." As in hæmophilia, however, this rule is by no means invariable. Stilling records inheritance from a grandfather through an unaffected

father. In Schoeler's case one out of five colour-blind sons had a
colour-blind daughter, and another had four colour-blind sons.
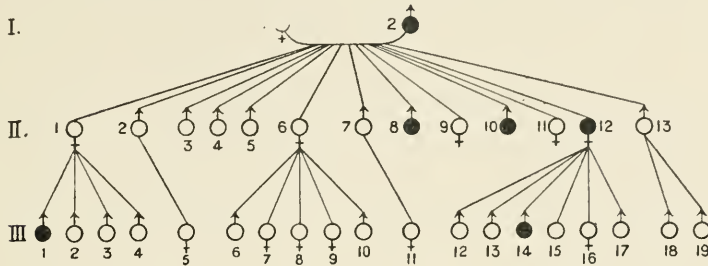
FIG. 890.—COLOUR-BLINDNESS.
Nettleship, T. O. S., xxvi.

Cunier published a remarkable family in which through five
generations only women, to the number of thirteen, were affected, but
the case is almost certainly not one of congenital colour-blindness.
Nettleship has recently published pedigrees containing colour-blind
women (Figs. 888—891).
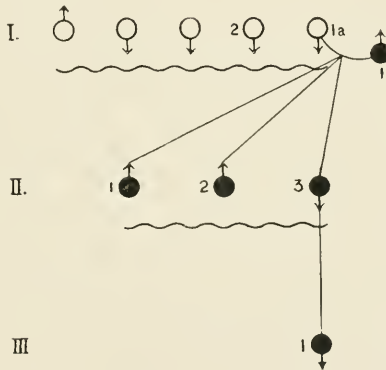
FIG. 891.—COLOUR-BLINDNESS.
Nettleship, T. O. S., xxvi.

EARLE.—Am. Jl. of Med. Sc., 1845. STILLING.—K. M. f. A., xiii, 1875, Beilageheft.
HORNER.—Mitt. a. d. ophth. Klinik, Zürich, 1876. RAEHLMANN.—A. f. O., xxii, 1, 1876.
HOLMGREN, COHN AND MAGNUS.—C. f. A., ii, 1878. SCHOELER.—Jahresbericht, Stockholm,
1878. BECKER.—A. f. O., xxv, 2, 1879. COHN.—Studien ü. d. angeb. Farbenblindheit,
Breslau, 1879. SCHMITZ.—C. f. A., iv, 1880. REPORT OF COMMITTEE ON COLOUR-BLIND-
NESS.—T. O. S., i, 1881. BOLLINGER.—v. Bischoff's Festschrift, 1882. GEISSLER.—Die
Farbenblindheit, Leipzig, 1882. HILBERT.—C. f. A., vii, 1883. KROLL.—C. f. A., xii, 1888.
MAUTHNER.—Farbenlehre, Wiesbaden, 1894. NORRIS AND OLIVER.—System, 1897.
PFLÜGER.—B. d. o. G., 1898. NETTLESHIP.—T. O. S., xxvi, 1906.

Nystagmus.—Congenital nystagmus, unassociated with other gross
disorder, has been seen in four generations (Owen), and often in three
(MacGillivray, Boulland, Audeoud). Inheritance may be discon-
tinuous. Burton saw in one generation only males, in another only

females affected. In these cases Friedreich's ataxia and spasmus nutans must be eliminated. Cases of albinism sometimes show only nystagmus without albinism in certain members of the family.

OWEN.—Ophth. Rev., i, 1882. BOULLAND, HERVOUET.—Rec. d'O., 1893. AUDEOUD.—Ann. d'Oc., cxiii, 1895. BURTON.—Lancet, 1895. MACGILLIVRAY.—Ophth. Rev., 1895.

Impairment of ocular mobility.—In 16 families with congenital impairment of ocular mobility, with or without ptosis, and excluding ptosis alone, in 3 there was collateral inheritance, in 7 two generations were affected, and in 4 three ; in 1 other branches of the pedigree were affected, in 1 there was consanguinity of parents. Collateral inheritance affects males (Vossius), and males and females (Guende, Kunn). The father never transmitted the disease to the daughter only, but twice to one son (Schiler, Pflüger), once to a son and a daughter (Rampoldi), once to a son and 2 daughters (Lawford). The mother transmitted the defect once to a son (Rohde, Uhthoff), once to a daughter (Harlan), once to 2 sons and a daughter (Heuck). In transmission through 3 generations it was three times from grandfather to father, once from grandfather to mother. The father transmitted the defect once to 1 son (Hirschberg), once to 4 sons (Gourfein), and once to 3 daughters (Günsburg). Gunn records 2 brothers with ptosis and almost complete immobility of the eyes ; there was consanguinity of the parents. Gazépy reports the defect in two branches of a family. In Beaumont's case of transmission of ophthalmoplegia externa to 14 persons through 4 generations it was in no case congenital.

HEUCK.—K. M. f. A., xvii, 1879. v. GRAEFE.—In G.-S., vi, 1880. HARLAN.—T. Am. O. S., 1885. HIRSCHBERG.—Neurol. Centralbl., 1885. LANDESBERG.—K. M. f. A., xxiv, 1886. LAWFORD.—T. O. S., vii, 1887. RAMPOLDI.—Ann. di Ott., xvi, 1887. ROHDE.—Dissertation, 1887. GÜNSBURG.—K. M. f. A., xxvii, 1889. VOSSIUS.—B. z. A., v, 1892. GUNN.—T. O. S., xiii, 1893. GAZÉPY.—A. d'O., xiv, 1894. GUENDE.—Rec. d'O., 1895. *KUNN.—B. z. A., xix, xxi, xxvi, 1895. PANAS.—Ann. d'Oc., cxvi, 1896; A. d'O., xvi, 1896. *WILBRAND AND SAENGER.—Neurologie des Auges, i, Wiesbaden, 1899. BEAUMONT.—T. O. S., xx, 1900.

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(Where there are several references the most important is printed in *italics*.)

GENERAL INDEX

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